A STUDY OF FIFTY SUSPECTED CASES OF CEREBRAL VENOUS THROMBOSIS IN ADULT WOMEN

Dissertation Submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

for

M.D. Degree in General Medicine

March 2008

DEPARTMENT OF GENERAL MEDICINE
COIMBATORE MEDICAL COLLEGE & HOSPITAL
COIMBATORE

CERTIFICATE

This is certify that the Dissertation entitled **"Study of Fifty Suspected Cases of Cerebral Venous Thrombosis in Adult Women",** herewith submitted by **Dr. Ramya. J ,** *M.D.,* Post Graduate in General Medicine , Coimbatore Medical College to the Tamilnadu Dr. *M.G.R.* Medical University is a record of a bonafide research work carried out by her under my guidance and supervision from Jan 2006 to Jun 2007.

Professor Dr. P. Jambulingam
Prof. and Unit Chief

Prof. Dr. K. Umakanthan
Prof. and Head
Department of Medicine

DEAN

DECLARATION

I solemnly declare that the Dissertation titled **"Study of Fifty Suspected Cases of Cerebral Venous Thrombosis in Adult Women",** was done by me at Coimbatore Medical College & Hospital during the period from Jan 2006 to Jun 2007 under the guidance and supervision of Prof. Dr. K. Umakanthan and Prof. Dr. P. Jambulingam.

This dissertation is submitted to the Tamilnadu Dr. *M.G.R.* Medical University towards the partial fulfillment of the requirement for the award of M.D. Degree (Branch *I*) in General Medicine.

Place : Coimbatore **Dr. J. Ramya**

Date :

ACKNOWLEDGEMENTS

I thank **Dr. T.P. Kalanidhi M.D.,** and **Dr. Hemalatha M.D.,** our beloved Deans for permitting me to conduct the study in this institution.

I express my sincere thanks and deep sense of gratitude to **Dr. K. Umakanthan M.D.,** Professor and Head, Department of Medicine for his able guidance and constant encouragement.

I acknowledge my deep sense of gratitude to **Dr. P. Jambulingam M.D.,** Professor of medicine and my Unit Chief for his valuable suggestions and guidance, without which it would have been

impossible to complete this work in the present form.

I am deeply indebted to **Dr. K. Govindarajan M.D., D.M.,** Prof. and head of Neurology for his support and constant encouragement.

I am very much obliged to **Dr. Ramakrishnan M.D., D.M.,** Asst. Prof. of Neurology for his help and guidance.

I thank **Dr. Kumar Natarajan M.D., Dr. P.Vishnuram, M.D., Dr. Rani, M.D.,** Asst. Professors of Medicine for their advice.

I acknowledge the co-operation of all the **Staff and Technicians** I am indebted to all the patients and their family members for their sincere cooperation without which this endeavour would not have been a success.

CONTENTS

Introduction

INTRODUCTION

Cerebral venous thrombosis is characterized by clinical pleomorphism and pathogenic variability. Though uncommon compared to arterial disease, it is an important consideration because of its potential morbidity.

Though recognized over 100 years, it has only recently come to be diagnosed frequently antemortom. This is partly due to greater awareness among physicians and neurologists and partly to improved non invasive imaging techniques especially Magnetic Resonance Venogram. Its clinical presentation is often dramatic, mimicking a variety of clinical conditions. It often afflicts young and middle aged patients, more commonly women .Studies have highlighted greater prevalence of disease in Indian population, more so in pregnancy and puerperium. Though earlier studies have reported higher mortality, recent studies have reported lesser mortality due to earlier diagnosis, increased awareness and management.

Though it occurs in a setting of a known predisposition to venous thrombosis, the proportion of unknown etiology remains higher. Since it

presents with a remarkably wide spectrum, it is often difficult to diagnose CVT on clinical grounds and requires neuroimaging for diagnosis. MRV has taken over cerebral angiography as the diagnostic modality of choice.

Management of CVT with anticoagulants especially heparin was a controversy in earlier studies , but recent studies have proved heparin to be both effective and safe even with haemorrhagic infarcts.

Outcome is unpredictable, mortality is reported to be lesser in recent series

This study is targeted to highlight the importance of recognizing and diagnosis of CVT at an early stage, to reduce morbidity and mortality of this potentially curable disease

Aim of the Study

AIM OF THE STUDY

I . To analyse the commonest clinical modes of presentation

II . To analyse the topography of involved venous sinuses in Magnetic resonance venogram.

III . To evaluate clinical outcome.

Review of Literature

REVIEW OF LITERATURE

HISTORY

The syndrome of intracranial venous and sinus thrombosis termed as Cerebral Venous Thrombosis was recognized in the early part of 18th century when Ribes [1] (1825) described in a 45 year old male , the clinical and autopsy spectrum of superior sagittal thrombosis. The first ever description of superior sagittal sinus thrombosis occurring during puerperium was by Abercrombie [2] in 1828. Kalabagh[3] in his monograph on CVT, stated that aseptic thrombosis is not an uncommon entity especially in puerperium and elderly. During the 1940s Sir Charles Symonds and Pardon Martin defined the clinical syndrome and provided basis for suspecting CVT as an antemortem diagnosis.

INCIDENCE

The exact incidence of CVT is still under debate because of scarcity of scientifically planned epidemiological studies in the available literature. With the advent of newer imaging techniques the prevalence of CVT is more common than reported previously[4,5,6].Aseptic CVT occurring in pregnancy and puerperium has been reported very frequently

from Indian population. CVT constitutes 10 -15 % of stroke according to Indian studies [7,8,9] especially in the young and was the commonest cause of stroke in pre-menopausal women. Prevalence in UK is 4/1000000/year. Prevalence of postpartum CVT in India is 4.5/1000/year[10,11]. In Indian population, mutiparas are affected more than primiparas in proportion of 4:1[12]. In 1995, Daif reported a frequency of 7/100000 in cases in Saudi Arabia. It has been estimated that prevalence rates of CVT in developing countries is approximately 10 times more than in developed countries. Currently aseptic CVT has replaced septic CVT as the commonest cause.

Scanning age related incidence graphs reveal three peaks as follows:[13,14]

1. Infants and children: Probably explained on basis of greater prevalence of dehydration associated diseases, malnutrition, CNS infections.
2. Young pre-menopausal women: Frequent use of oral contraceptive pills in developed countries is an important etiological factor, while in developing countries pregnancy and puerperium is the commonest cause.

MORTALITY

According to British Registrar General [15], average mortality in UK was 0.4/1000000/year i.e 10%. In early literature mortality ranged between 30 -50 % [16 ,17,18] whereas it is between 10 -15 %[19 ,20, 21, 22] in recent series. More recently a Portuguese study group prospectively analyzed 91 consequently admitted patients from 1995-1998, of patients analyzed 7% died in acute phase, 1% died during one year follow up, 82% recovered completely. 59% developed thrombotic events, 10% seizures, 11% complained of severe head ache.

ANATOMY[23,24]

Knowledge of the venous anatomy is essential to understand the pathogenesis, clinical manifestations, prognosis in intracranial sino-venous disease. The cerebral venous system is unique in being valveless, mostly trabaculated, low pressure system. The cerebral venous system consists of

(1) Dural venous sinuses

(2) Cerebral veins

CEREBRAL VEINS : Divided into superficial/external group and deep/internal group.

SUPERFICIAL CEREBRAL VEINS : Arise from cortex and medullary substance of hemisphere.

1. Superior Cerebral Veins : 8-12 in number, drain superior, medial, lateral surfaces of cerebral hemisphere above sylvian fissure, pierce arachnoid membrane and terminate in superior sagittal sinus.

2. Middle Cerebral Veins : Traverse sylvian fissure, drains insula and opercular region and terminate in cavernous or spenoparietal sinus. It is connected to superior sagittal sinus by the Great anastomotic vein of Trolard, and with tranverse sinus by anastomotic vein of Labbe.

3. Inferior Cerebral Veins : Many in number, divided into orbital and temporal veins. Orbital veins terminate into superior cerebral veins or superior sagittal sinus.

Temporal veins terminate into cavernous, transverse, superior petrosal sinus through middle cerebral veins.

DEEP CEREBRAL VEIN : Drain the interior of cerebral hemispheres.

1. Choroidal Veins : Runs entire length of choroids plexus, and receives branches from hippocampus, fornix and corpus collosum. It joins with the terminal vein to form the Internal cerebral vein.

2. Terminal Vein : Runs in the groove between caudate nucleus and thalamus. It received many tributaries from these structures as well as from the internal capsules.

The two internal cerebral veins join to form the Great cerebral vein of Galen, that terminates in the straight sinus.

3. Basal vein of Rosenthal : Formed by the union of anterior cerebral vein, deep middle cerebral vein and inferior striate vein. It receives tributaries from cingulate gyrus, anterior part of corpus collosum, orbital surface of frontal lobe, olfactory groove, optic chiasm, hypophysis, cerebral peduncle, inter peduncular fossa, mid brain.

VENOUS SINUSES

1. Superior Sagittal Sinus : Occupies the falx cerebri. Starts anteriorly in the crista galli and ends in the internal occipital protuberance by turning to the right side and continuing as the right transverse sinus. It receives the superior cerebral veins and the parietal emissary veins.

2. Inferior Sagittal Sinus : Situated in the posterior 2/3 of falx cerebri. It joins the great cerebral vein to form the straight sinus that eventually ends in the left transverse sinus.

3. Straight Sinus : Formed by the union of the inferior sagittal sinus and the great vein of galen to continue as the left transverse sinus. At the termination of the great cerebral vein into the sinus, there exists a ball valve mechanism formed by a sinusoidal plexus of blood vessels which regulates the secretion of cerebrospinal fluid.

4. Transverse Sinus : Begins at the confluence of sinus (Torcula Heterophili) to pass laterally and forward in the margin of tentorium cerebelli to the postero inferior angle of the parietal bone and passes down as a sigmoid sinus. It receives blood from the superior petrosal sinus, inferior cerebral veins, inferior cerebellar veins.

5. Sigmoid Sinus : It is the direct continuation of the tranverse sinus and is ‘S’ shaped. It extends from the postero inferior angle of the parietal bone to the posterior part of the jugular foramen where it continues as the superior bulb of internal jugular vein. Its tributaries are the mastoid veins, cerebellar veins, internal auditory vein.

6. Cavernous Sinus : It is a large venous space between the two layers of the duramater situated in the middle cranial fossa, one on either side of the body of the sphenoid bone. It contains a cranial nerves III, IV, V,internal carotid artery, and VI nerve. Its tributaries are inferior ophthalmic vein, central vein of the retina, superficial middle cerebral vein, inferior cerebral veins, spenoparietal sinus, and frontal trunk of middle meningeal vein. It drains into the transverse sinus through superior petrosal sinus, into the internal jugular vein through inferior petrosal sinus, into the pterygoid plexus of veins through the emissary veins, into the facial vein through superior ophthalmic vein.

7. Superior Petrosal Sinuses : Lie in the anterior part of the attached margin of tentorium cerebelli along the upper border of the petrous temporal bones.These sinuses drain the cavernous sinus into the transverse sinus.

8. Inferior Petrosal Sinuses : Lie in the petro-occipital fissures and drain the cavernous sinus into the internal jugular vein.

9. Spenoparietal Sinus : Receive the frontal trunk of middle meningeal vein and drain into the anterior; part of the cavernous sinus.

FUNCTIONAL ANATOMY[23]

1. The cerebral veins and sinuses have neither any valves nor any tunica muscularis. Absence of valves permits blood flow in various directions , while absence of tunica muscularis permits the veins to remain dilated.
2. Intercommunication between the various venous sinuses provide alternative routes by which blood from dural sinuses may flow if normal drainage is blocked.
3. Venous sinuses are located between the two rigid layers of dura mater. This prevents their compression when intra cranial pressure rises.
4. Emissary veins from scalp, face, ears, Para nasal sinuses etc and the diploic and meningial vein drain directly into venous sinuses and thus facilitates spread of infections. For example, superior sagittal

sinus thrombosis in scalp infections, cavernous sinus thrombosis in facial infections and otitis in lateral sinus thrombosis.

5. Superficial cortical veins drain into the superior sagittal sinus against the blood flow in the sinus, thus causing a turbulence in the blood stream that is further aggravated by the presence of fibrous septa in the inferior angle of sinus. This explains the greater prevalence of superior sagittal sinus thrombosis.
6. Arachnoid villi are located in the walls of superior sagittal sinus and drain the cerebrospinal fluid into the sinus. So any thrombosis in the sinus will block the villi and raise the intra cranial tension.
7. The deep cortical veins form venous circle around the mid brain. And get engorged in superior sagittal sinus thrombosis.

ETIOLOGY

More than 100 causes of CVT have been recorded in scientific literature [21.] However with extensive investigation no cause is identified in 25% cases.[24]

CVT can develop because of :

1. Changes in vessel wall- e.g. Malignancy, infection.
2. Changes in blood flow-e.g. Stasis.
3. Changes in blood coagulability : e.g. anti-phospholipid syndrome.

CAUSES :

I. Hyper coagulable causes: Pregnancy, Puerperium [25,26,27], APLA [28,29] Anti-thrombin III deficiency [29] ,Protein C and S alteration [29,30], Factor V Leiden mutation [29], paroxysmal nocturnal haemoglobinuria [31], Thrombotic thrombocytopenic purpura, Polycythemia [19], Sickle cell diseases [32], Hyper homocystinemia[33], nephrotic syndrome[34], Leukemias[35],multiple myeloma, waldenstroms macroglobulinemia, Budd chiari, DIVC.

II. Changes in vessel wall: Malignancy [36,37,38], infections[39, 40, 41, 42,43,44] (Otitis, Facial, Nasolabial, Meningial, Post traumatic)

III. Changes in blood flow: Dehydration, congestive cardiac failure.

IV. Drugs [45]: Oral contraceptives [46 47,] steroids [48], epsilon amino caproic acid, L-asparaginase, ecstasy [49,]heparin [50].

V. Others: Inflammatory bowel disease [51], cirrhosis liver ,sarcoidosis [52,] connective tissue disorder [53 54 55].

VI. Unknown cause.

POST INFECTIVE/SEPTIC CVT [19, 26 ,27]

Was the commonest cause of CVT in pre antibiotic era. The cavernous sinus, lateral sinus, and superior sagittal sinus were commonly involved in order.

CVT IN PREMENOPAUSAL WOMEN[56, 57, 58 ,59 ,60, 61]

CVT in pregnancy and puerperium, is the commonest cause of CVT in developing countries and is frequently reported from the Indian subcontinent [10.] It usually occurs in the last trimester of pregnancy and in the puerperium usually within the first three weeks [62].

The contributing factor is hyper coagulability that occurs due to [63] :

1. Increased clotting factors – I, VII, VIII, X.
2. Decrease coagulant inhibitors like protein S and C[64.]
3. Increased ability to neutralize heparin.
4. Factor V Leiden mutation[65].
5. Fluid restriction following delivery leading to dehydration and changes in blood flow.
6. Anemia of pregnancy[10].
7. Raised serum triglycerides, decreased fibrinolytic activity[66,67], increased platelet count and their adhesiveness[68] have been observed but statistical correlation studies have disproved their role.

Earlier observations of venous stasis, puerperal sepsis and paradoxical embolism through the vertebral plexus as the contributing factors for CVT in pregnancy and puerperium have not been established.

Post puerperal CVT is found to be more commoner than post gestation CVT in Indian studies which also found it to be more prevalence among multiparas than primiparas in the ratio of 4:1.

CLINICAL FEATURES

Varied presentation, determined by:

1. Underlying venous system involved
2. Mode of onset-acute / subacute / chronic
3. Time if interval between disease onset and clinical presentation
4. Nature of the primary disease giving rise to CVT

AGE : In1992, Ameri and Bousser reported a uniform age distribution among men with CVT, while most women were reported in the age group of 20-30 years, this may be related to use of oral contraceptives.

SEX : CVT is believed to be more common among women than men in a series of 110 cases, Ameri detected a female-male ratio of 1.29:1.

SYMPTOMS :

1. HEADACHE : is almost the universal symptom of primary CVT. It is the most frequent and often the presenting symptom. It may be unilateral or bilateral and often involves the frontal or temporal regions. Vomitting

usually accompanies the headache and is more common among patients with raised intracranial tension. The headache results from raised intracranial tension and involvement of pain sensitive sinuses. Once anticoagulation is begun the headache improves markedly. Thunderclap headache resembling subarachnoid hemorrhage is a rare presentation [69]. Patients presenting with headache as the only sign is rare and usually have accompanying neurological signs [70.]

2. FEVER : may be prominent in patients with septic CVT ,but may occur due to an aseptic thrombotic process.

3. SEIZURES : are very common and often begins early ,may be focal or generalized and present in 50% of cases[60].Their early appearance is a sign of bad prognosis. The seizures may be recurrent and may be followed by aphasia and hemiparesis.

4. FOCAL NEUROLOGICAL DEFICITS : comprises hemiparesis usually with facial sparing ,as facial area is drained by slyvian vein. The lower limb is affected more often than upper limbs[58]. In some cases of superior saggital sinus the contralateral cortical veins may be involved giving rise to contralateral paraparesis or paraplegia [59,61].

Cortical deficits like aphasia , apraxia, agnosia are not uncommon and are fleeting in nature.

5. CRANIAL NERVE SYMPTOMS : usually due to raised intracranial tension and septic CVT involving cavernous sinus. Obscuration of vision, double vision, facial weakness, deafness, tinnitus may be the presenting complaints.

6. COGNITIVE DYSFUNCTIONS: drowsiness, irritability, confusion, varying degrees of coma.

7. OTHERS : psychiatric disturbances, akinetic mutism, fluctuating blood pressure.

SIGNS :

1. Variable conscious state-fully conscious to coma.
2. SUPERIOR SAGGITAL / LONGITUDINAL SINUS THROMBOSIS [60] : Headache, vomiting, papilloedema , hemiplegia, cortical type paraplegia [3.]

3. LATERAL SINUS THROMBOSIS (transverse and sigmoid sinuses) : Fever, headache ,mastoid swelling, lower cranial nerve involvement if thrombus involves jugular foramen.

4. CAVERNOUS SINUS THROMBOSIS [27]: As it is usually post infective ,its incidence has decreased with advent of antibiotics. Recently there has been a spurt in its incidence due to emergence of drug resistant organisms and immunosupressive disorders .Sources of pyogenic organisms are nasolabial territories and para nasal sinus.

   Fever, retro orbital pain, chemosis, proptosis, orbital congestion, III,IV,VI nerve involvement causing ophthalmoplegia, blindness due to optic nerve involvement, papilloedema, hemiparesis due to internal carotid artery involvement.

5. CEREBRAL VEIN THROMBOSIS: Superficial vein thrombosis : partial seizures, hemiparesis , aphasia. Its involvement alone without extension into dural sinuses is rare and lacks features of raised intracranial tension[71].

   Deep cerebral vein thrombosis [72]: involvement is rare ,accompanied by impaired consciousness, abnormalities in eye movements ,papillary reaction, fluctuating blood pressure, quadriparesis.

The cerebellar veins [73], may occasionally be involved in relation to middle ear infections leading to slowly evolving syndrome of vertigo, vomiting and ataxia.

6. **PUERPERAL CVT:** Depending on the venous sinus involved, clinical picture is usually of headache, seizures, focal deficits, papilloedema . Sometimes patients may present with headache and papilloedema without focal deficits simulating a brain tumour-pseudo tumour cerebri [35] The onset of symptoms in 80-90% of CVT is within the 1^{st}-2^{nd} week of delivery. Presentation 3months following delivery has been observed in studies by Nagpal in 1983. A large number of affected persons are multiparous [12] according to Indian studies (Banerjee, 1973; Bansal,1980; Srinivasan, 1983). According to a U.S. based survey of puerperal CVT [63], a significant risk was with cesarean section, increasing maternal age, hyperemesis ,maternal hypertension, intercurrent infections .The association of hypertension with puerperal CVT has not been reported with other studies.

Based on the experience of dealing with large number of cases of CVT Indian studies have grouped clinical pattern of CVT as follows:

GROUP-I : Meningo encephalitic type :headache ,fever ,seizures ,altered sensorium, focal deficits, meningeal signs.

GROUP-II : Acute fulminant type :status epilepticus, coma.

GROUP-III : Pseudo tumour type : headache ,vomiting , papilloedema.

GROUP-IV : Neuropsychiatric type : abnormal behaviour, with or without features of raised intracranial tension.

In a recent Dutch European study [22], the most frequent symptoms and signs were :

1. Headache - 95%
2. Papilloedema - 49%
3. Seizures - 47%
4. Motor/sensory deficits - 34%
5. Changes in consciousness - 30%
6. Dysphasias - 12%
7. Cranial nerve palsies - 12%
8. Nystagmus - 2%
9. Deafness - 2%

PATHOLOGY [74] : findings are determined by following factors:

1. Underlying disease pathology
2. Nature of venous sinus/cerebral vein involved
3. Interval between onset and pathological examination

Cortical vein thrombosis :

Presents as a cord like swelling with very minimal hemorrhagic infarction of the brain. This is due to the presence of frequent intercommunications between various cortical veins and sinuses.

Superior sagittal sinus thrombosis:

Sinus appears distended and blue. Cortical veins also get ruptured giving rise to intracerebral hemorrhage. In an occasional case, hemorrhagic infarction may appear on the other side due go occlusion of opposite cortical vein.

Deep cerebral vein thrombosis :

White matter area of brain gets involved , especially the regions of basal ganglia and thalamus. As times progresses the thrombus gets recanalised, organized and may even disappear in majority .Cerebral edema due to venous flow obstruction may lead to transtentorial herniation with notching of uncus.

COMPLICATIONS :

1. Increasing venous congestion raises cerebrospinal pressure if collateral drainage is insufficient [75].
2. Parenchymal edema with venous infarction, hemorrhage complicates 50% cases [24].
3. Seizures may persist requiring continued anti epileptics.
5. Pulmonary embolism is uncommon but carries a poor prognosis. A review of literature between 1942-1990 revealed in 11.3% cases of venous sinus thrombosis was associated with pulmonary embolism and in these cases the overall mortality was 95.6%, far higher than those without embolism[76].
6. Hypopituitarism may result from cavernous sinus thrombosis [77].
7. Dural sinus thrombosis was associated has been cited by several authors as an aetiological association with dural A-V fistulas, although it may be difficult if the thrombosis was the primary or secondary event [78].

DIAGNOSIS

Requires a high index of suspicion

Objectives of investigation are:

1. Diagnosis of cerebral vein or sinus thrombosis.
2. Identification of the involved sinus or vein involved.
3. Identification of the underlying pathogenic factors.

I. C.T.SCAN BRAIN[79 ,80]

It is often the first investigation obtained. It may show both direct and indirect signs of cerebral venous thrombosis .It is also helpful to rule out other causes of patients clinical condition like neoplasm, abscess, arterial stroke.C.T.brain may be normal in 20% cases[16].

The direct signs are due to thrombosis of sinus or vein , the indirect signs are due to secondary effects of thrombosis on parenchyma [81].

A. DIRECT SIGNS : the two signs reported as pathognomonic of C.V.T ,the cord and delta signs are found in only minority of cases [82].

Plain C.T

CORD SIGN [83 ,84] **:** represents the thrombosed cortical vein. It appears as a linear hypodense streak in location of involved vein. It represents acute or new thrombus ,thus seldom seen in chronic cases.

DENSE TRIANGLE SIGN [83 ,84] **:** triangular area of increased density along course of superior sagittal sinus and represents opacification by freshly coagulated blood. It is rare and usually seen in the first two weeks of the disease.

Contrast C.T

DELTA /EMPTY TRIANGLE SIGN[16] **:** it is a triangular rim of contrast surrounding a clot within the superior sagittal sinus ,giving appearance of Greek letter delta. It is the most frequent sign present in 30% of cases .This sign appears after first 3-5 days of disease onset .Early division of superior sagittal sinus can be some times be responsible for false delta sign.

B. INDIRECT SIGNS : they are frequent and include :

1. Intense contrast enhancement of falx and tentorium, a sign of straight and superior sagittal sinus thrombosis. Seen in 20% cases and indicates venous stasis and hyperemia of the duramater.

2. Single or multiple areas of intense gyral enhancement.
3. Brain oedema-may be unilateral or bilateral. Seen in 25-50% cases. It is characterized by white matter hypodensity on C.T, compressed ventricles ,obliterated cisterns, effaced sulci.
4. Venous infarcts : seen in 10-15% cases .Can be unilateral or bilateral, hemorrhagic or non hemorrhagic.

   Hemorrhagic infarct appears as hyperdensities within hypodense infracted zone plain C.T. Both petechial hemorrhages as well as large hematomas may be encountered. Superior sagittal sinus produces frontal or parietal infarct. Lateral sinus thrombosis produces temporal lobe infarct. Diencephalic or basal ganglionic in infarcts deep cerebral vein thrombosis.

   Non hemorrhagic infarct appears as focal areas of hypodensities on C.T.

   Venous infarcts are differenciated from arterial infarct as follows:

   *More often hemorrhagic

   *Do not confirm to any arterial territory.

   *Have ill defined margins.

   *Have greater mass effect for the size of infarct.
5. Hydrocephalus:due to raised intra cranial tension in superior sagittal sinus thrombosis.

II. MAGNETIC RESONANCE IMAGING / MAGNETIC RESONANCE VENOGRAM [85]

In 20% cases C.T. scan can be normal. The use of MRV has led to early, accurate and non invasive diagnosis of C.V.T.MRV has replaced invasive cerebral angiography as the investigation of choice for CVT [86].

Findings : depends on the age of the thrombus

*If the venous sinus or cerebral veins are acutely thrombosed , there is no signal or flow at the anatomical site of the vein .The absence of flow can also result from anatomical absence of vein , so it is necessary to perform axial/sagittal T1/T2 weighted MRI.

*On T1 and T2 weighted images, the acute thrombus appears isointense onT1 and hypointense on T2.Few days later the thrombus becomes hyperintense on T1 and T2 weighted images.

*Two weeks later the thrombus reduces in size with recanalization and resumption of normal flow void.

ADVANTAGES:

*Sensitive to blood flow changes and helps visualize the thrombus.

*More sensitive than C.T.in detecting parenchymal changes particularly microhemorrhages ,considered hall mark of venous infarct [87,75].

*Demonstrate underlying cause such as tumours or unsuspected mastoiditis.

DISADVANTAGES :

Difficulty in diagnosis may arise due to normal anatomical variants [88].

Commonest venous channels involved in CVT in MRV are [85]:

1. Superior sagittal sinus thrombosis - 72%
2. Transverse sinus thrombosis - 70%
3. Straight sinus - 14%
4. Deep sinus - 8%
5. Cavernous sinus - 3%.

III. CEREBRAL ANGIOGRAPHY

Invasive test, was regarded as the investigation of choice for CVT, now replaced by MRI/MRV.A four vessel angiography with venous phase films taken at intervals of 5-12 seconds of contrast injection. Frontal, lateral, oblique views are taken in order to visualize the entire venous system. Delayed venous films may be necessary to allow for slow venous filling in patients with raised intracranial tension .Angiography becomes necessary if thrombolytic therapy is considered.

FINDINGS :

1. Failure to demonstrate all or part of sinus/vein or localized irregularity within its wall.
2. Non filling of cortical veins
3. Focal or diffuse slowing of circulation and stagnation of coagulated blood in capillary or venous phase.
4. Corkscrew vessels that do not reach cortical surface due to dilatation of anastomotic vein over cortex.

5. Parenchymal changes include:

-Focal or diffuse brain swelling with sulcal effacement and chinked ventricles in severe cases.

-Intracranial hemorrhage as a result of intra luminal pressure exceeding the structural limit of vessel wall

-Ventriculomegaly due to decreased cerebrospinal fluid, a consequence of increased resistance of CSF resorption by the arachnoid granulations.

While reviewing angiogram films following points to be remembered :

*Venogram films may be normal due to well developed collaterals

*Recanalisation of sinus may also yield false negative angiogram.

*The Vein of Galen may not be visualized in patients with raised intracranial tension.

*Non visualisation of sinus may be due to anatomical variation

*Narrow anterior part of superior sagittal sinus is often poorly visualized at angiography and its isolated lack of filling is not sufficient to indicate thrombosis.

*Cavernous sinus are rarely visualized on angiogram.

*Similarly an isolated lack of filling of transverse portion of the left lateral sinus is more suggestive of hypoplasia than thrombosis.In one

study transverse portion of lateral sinus was not visualized in 14% cases on left and 33% cases on right.

IV. RADIONUCLIDE SCANNING [89]

Dynamic radionuclide scanning has been utilized for visualization of venous dural sinuses, but lacks sensitivity and specificity.

V. OTHER INVESTIGATIONS

1. CSF examination [19] : not of diagnostic use .It is performed in suspected meningitis .It may reveal non specific changes like raised proteins ,cells and pressure.
2. Appropriate tests towards aetiogenesis like :

   *Complete blood count-may reveal anemia,polycythemia,leucocytosis,thrombocytopenia.

   *Lupus anticoagulant and anti cardiolipin antibodies.

   *Protein C and S,Antithrombin III, FactorV Leiden mutation

   *Sickle cell preparation

   *ESR, ANA tests in connective tissue disorder

   *ANCA in wegeners granulomatosis

   *Urine for protein

   *Liver function tests

*D-dimer [90]-in a prospective study of 54 patients with headache suggestive of CVT Lalive found 12 had CVT,10 had D-dimer of >500ng/ml. The 2 patients had chronic headache >30days. D-dimers were positively correlated with the extend of thrombus and negatively correlated with duration of symptoms.

*EEG-abnormal in 75%cases.Changes are nonspecific and include:

-Focal or diffuse hemispheric slowing.

-Focal or generalised epileptic activity

EEG may not add to the diagnosis but may guide seizure management in long run.

MANAGEMENT :

Aim of treatment is to:

1. Provide symptomatic relief to patients.
2. To prevent further extension and recurrence of thrombus.

Treatment is based on:

1. General therapy
2. Symptomatic therapy
3. Specific therapy.

GENERAL MEASURES :

Include routine measures for care of comatose patient and includes:

*Maintenance of airway patency.

*Maintenance of fluid and electrolyte balance.

*Prevention of infections and gastric ulcers.

SYMPTOMATIC TREATMENT :

1. **Seizures :** antiepileptics are obviously indicated in patients with clinical seizures. However controversy exists regarding:

   *Prophylactic use of anticonvulsants in all patients with CVT.

   *The duration of therapy, once antiepileptics are started-may range from 2-5 years. Ideally a one year seizure free interval is reasonable.

2. **Cerebral oedema :** cytotoxic edema results due to alteration in biochemical mechanism at cellular level. It is followed by vasogenic edema in 24-48 hours .Measures to control cerebral edema and raised intra cranial tension are :

   *Head end elevation to 30 degrees to promote venous drainage.

   *Mannitol(20%)-in dose of 0.25-1g/kg i.v over period of 10minutes

   *Dexamethasone therapy is of unproven efficacy but can be given in a dose of 4mg every 6 hours.It primarily reduces vasogenic edema.

   *Hyperventilation to bring partial pressure of carbon dioxide to 25-30mmhg to reduce intracranial pressure in life threatening situations.

*Other agents/ measures used are-glycerol, acetazolamide, shunting and even barbiturate induced coma.

*The choice among these methods depends on the clinical situation. If the level of sensorium deteriorates or if the headache is severe mannitol is preferred.

*Surgical decompression will be required in cases of continuing deterioration inspite of maximum medical management.

3. **Infections :** appropriate antibiotics.

SPECIFIC THERAPY :

I. ANTICOAGULANT THERAPY: with heparin was first advocated for venous thrombosis 50years ago [32]. Its use in CVT has been debated for long time. In the past , the main arguments cited against its use were presence of hemorrhagic infarction, subarachnoid hemorrhage. Scientifically planned studies have generated enough evidence in favour of heparin use and thus has emerged as a useful drug in management of CVT [91, 92 ,93]. In the past anticoagulation was mainly reserved for patients with coexistent deep vein thrombosis, pulmonary embolism and prothrombotic states. Currently anticoagulation with heparin either intravenous/subcutaneous is indicated for all cases of CVT with or without hemorrhagic infarcts provided there is no general contra indications for its use [94 95]. Neonatal CVT is the only situation in which heparin has not been shown to improve the outcome and most authors do

not recommend its use in this situation (Nagaraja, Sarma 2002). Heparin reduces both morbidity and mortality in CVT[96, 97]. Anticoagulants are used to prevent propagation of the clot to more extensive areas of cerebral venous system and thereby prevent progression of infarction.

A. HEPARIN : Increases the action of antithrombin III, leading to inactivation of coagulation factors thrombin, factor Xa and IXa.

DOSE : Most studies have used higher doses of heparin.

The dose of heparin used should result in aPTT twice of the baseline. Both low molecular and unfractionated heparin are equally efficacious. Heparin is given parenterally for 2 weeks followed by oral anticoagulants for 3-6 months. Unfractionated heparin can be given either by subcutaneous or continuous IV infusion.

The results of two prospective trials conducted at the National institute of mental health and neurosciences support the use of low dose of heparin-15,000u/day subcutaneously in three divided doses in puerperal CVT.

Dose of continuous infusion is as follows :

80u/kg body weight bolus, 18u/kg/hr IV; aPTT checked in six hours and every six hours after any dosage change, adjust according to following parameters.

aPTT <1.2 times control – 80u/kg/hr; increase by 4u/kg/hr

aPTT 1.2 to 1.5	-	40u/kg/hr; increase by 2u/kg/hr
aPTT 1.5 to 2.3	-	no change
aPTT 2.3 to 3	-	decrease by 2u/kg/hr
aPTT > 3	-	hold infusion for 1hr, decrease by 3u/kg/hr

A more recent multicentric double blind placebo controlled trial of Nadroparin, a low molecular weight heparin by de Bruijn and Stam (1999) randomized 59 patients to receive Nadroparin and placebo for three weeks followed by oral anticoagulation for 3 months. After 12 weeks, 13% of Nadroparin and 21% in placebo group had poor outcome.

Contraindications for heparin use includes-documented hypersensitivity, active bleeding, coagulopathies, endocarditis, hemophilia, thrombocytopenia.

B. WARFARIN : interferes with the action of vitamin K, a cofactor essential for converting precursor proteins into factors 2,7,9,10. Does not

affect activity of coagulant factors synthesized prior to exposure. It used for 3-6 months following parenteral heparin.

Dose is 5mg/day, dose adjusted by monitoring prothrombin time and INR. Target INR is 2.5. Patients are then reevaluated at end of six months with magnetic resonance venogram to see if flow has been established.

Contraindications in documented hypersensitivity, pregnancy, active bleeding, vitamin K deficiency.

Prolonged anticoagulation therapy may be required in refractory cases or those with underlying prothrombotic state. In patients with previous post partum CVT, the use of prophylactic heparin in the subsequent postpartum period is not warranted.

II. THROMBOLYTICS : use of systemic anticoagulants for sinus trombosis was first used 30 years ago using Urokinase [98].There followed a series of open case studies using local infusions either through a frontal burr hole or more usually selective catheterization [99].

Various techniques of achieving thrombolysis include pharmacotherapy with urokinase or rtPA, mechanical thrombolysis, rheolytic thrombectomy.

Recombinant tissue plasminogen activator (rtPA/ALTEPLASE), has theoretical advantage in mechanism of action over Urokinase [21].Two series totaling 21 patients have been reported in literature of its use in dural sinus thrombosis [100 ,101], administered through jugular catheter. In most cases outcome was good .Local rtPA carries undoubted risk of increased symptomatic hemorrhage.

Thrombolysis causes clot lysis. All agents are directly instilled into the sinus at time of surgery or use of microcatheters to reach venous sinuses.

1. ALTEPLASE : converts plasminogen into plasmin, which degrades fibrin, fibrinogen, factor V and VIII. Dose is 1mg/cm infused through venous catheter, then 1-2mg/hour.

2. UROKINASE : dose is 2,50,000u/hour; additional doses of 50,000u;total dose 1,000,000.over 2hours.

3. STREPTOKINASE : dose is 1000-3000u/kg bolus,1000-1500u/kg/hour.

Contraindications to the above thrombolytics are : documented hypersensitivity, active bleeding ,severe hypertension ,trauma/surgery in previous 10 days, coagulopathy, endocarditis etc.

Rheolytic thrombectomy makes use of Bernoulli effect to create a vacuum that fragments and aspirates the thrombus. This has the added advantage of lack of hemorrhagic complications and potential for use even in hemorrhagic CVT.

Further studies on thrombolytics are in progress [102].

III. ANTIPLATELET AGENTS : have been advocated but never been systematically studied.

IV. SURGERY : surgical decompressive craniotomy, thrombectomy and evacuation of intracerebral hematoma need further evaluation before it is recommended as a safe procedure[103] .Out comes are usually poor [104].

In conclusion, patients need to be treated with heparin, even in those with pre existing hemorrhage .Failure to respond to therapy indicated by deepening coma or CNS deficits despite adequate heparinization should be treated with local thrombolytics.

PROGNOSIS : highly variable.

1. Poor Outcome : a recent study Dutch venous sinus thrombosis group looked at the prognostic factors in a series of 59 cases [22],poor outcomes was associated with:

*Very young/advanced age.

*Patients presenting with coma or rapid clinical course.

*Papilloedema

* Diagnostic delay>10days

*Intracerebral hemorrhage

*Involvement of deep cerebral and, cerebellar veins, straight sinus

Other studies, have suggested poor outcomes with the above parameters and others such as:[105 ,106]

*Uncontrolled seizures

*Pulmonary embolism

*Infections or malignant etiology.

2. Good outcome :

*Isolated intracranial hypertension at presentation

*Delta sign(perhaps due to early diagnosis)

Follow up in Dutch group found that in one year [107]:

35%had cognitive impairment, 6%were dependent, 40% had symptoms that led to restriction of life style,40%could not resume their previous level of economic activity. This suggests that there may be more morbidity after CVT than previously reported, interestingly there was no significant effect of treatment on this later outcome data.

RECURRENCE

Little is known about the risk of recurrence, although one study reports a risk as high as 12% [106]. Patients also have increased risk of (14%) venous thrombosis elsewhere in the body. Studies following recanalisation of venous sinus have shown that it can be incomplete in certain cases [108] .Raised intracranial pressure may persist following acute presentation of thrombus[75]. One study has shown that thus results in number of significant cognitive or visual disturbances[109].

Materials and Methods

MATERIALS AND METHODS

A prospective study, involving a series of 50 women presenting with clinical symptoms suspicious of cerebral venous thrombosis admitted at Coimbatore Medical College Hospital over a period of 1 ½ years – from January 2006 - June 2007 was analysed, patients were followed up for three months following discharge at Medical OPD.

A detailed history with respect to presenting complaints was taken. History of fever, bedridden state, dehydration, thrombotic and haemorrhagic tendencies, recurrent pregnancy loss were noted. Obstetric history was also taken. Patients vital parameters were assessed. General physical examination, CNS and other systemic examinations were done in the conventional way.

All patients underwent laboratory testing with complete blood count with peripheral smear , blood urea , serum creatinine , liver function tests , coagulation profile (PT, APTT) , serology for VDRL and HIV , CSF studies unless contraindicated. All findings were recorded in the proforma.

Neuroimaging studies were carried out in all patients, initially with cranial CT to rule out other causes of patients presenting complaints and to note any changes of CVT. All patients were further assessed with MRV.

Detailed analysis of the data was done with a view to find out the commonest clinical mode of presentation, topography of involved venous sinuses in the magnetic resonance venogram. All patients were treated with anticoagulant unfractionated Heparin 5000 units intravenously 8^{th} hourly and outcome at discharge was assessed as good (when alive with full functional recovery) and poor (death, partial functional recovery).

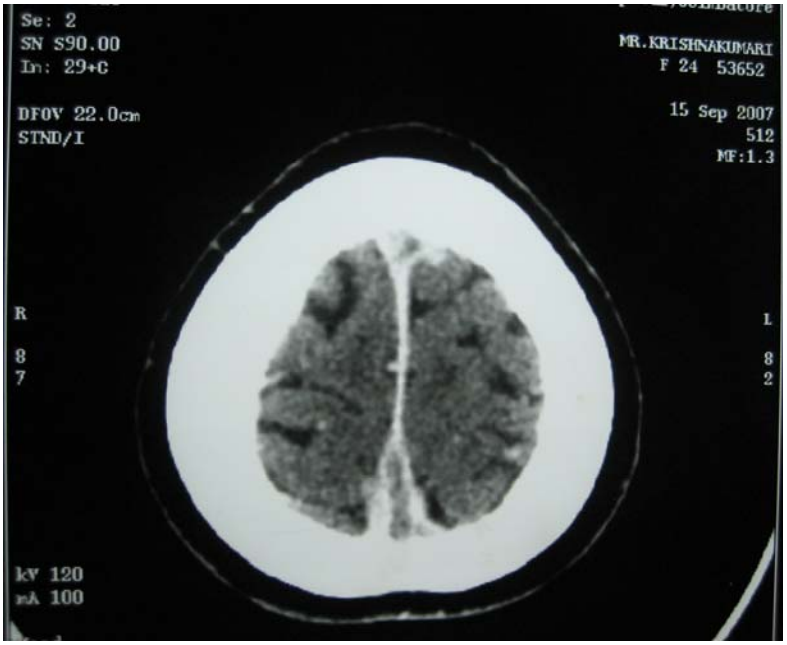
INCLUSION CRITERIA

1) Female patients
2) Age more than 13 years with clinical symptoms suggestive of cerebral venous thrombosis.

EXCLUSION CRITERIA

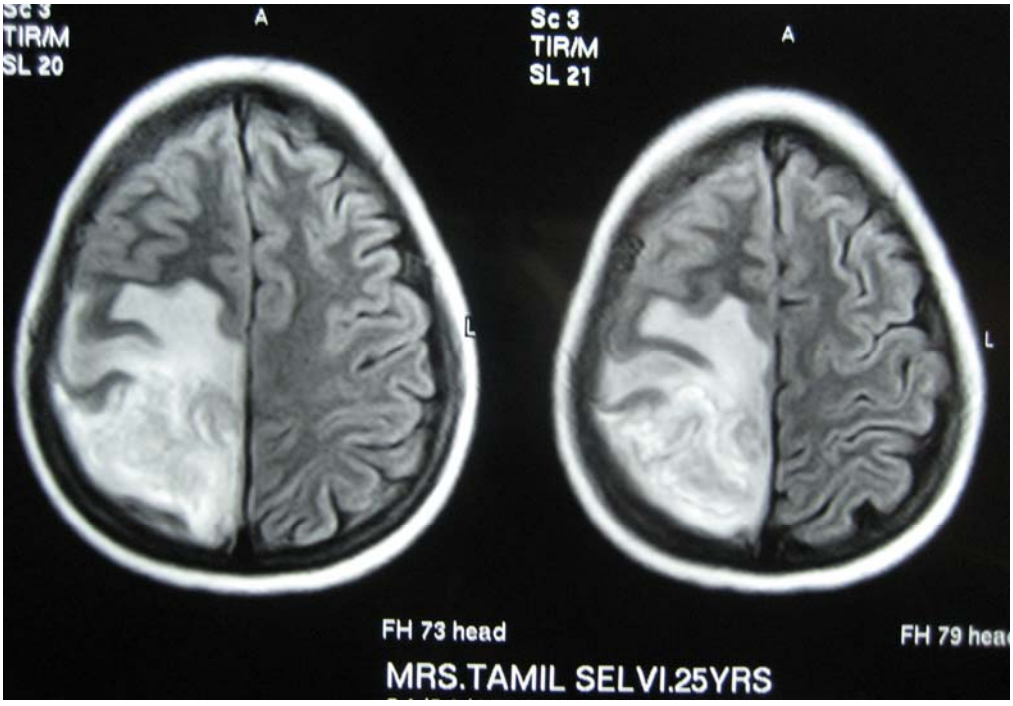
1) Female patients of age < 13 years
2) All cases with head injury, neoplastic brain diseases

CT CONTRAST
DELTA SIGN

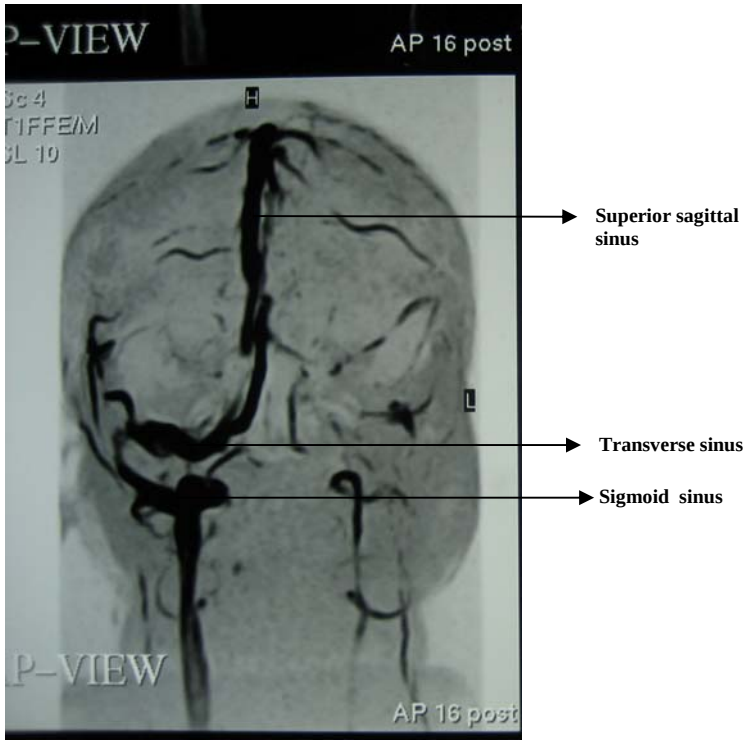


CT BRAIN - PLAIN
LEFT PARIETO TEMPORAL HEMORRHAGIC VENOUS INFARCT

MRV
RIGHT PARIETO TEMPORAL HEMORRHAGIC VENOUS INFARCT



MRV
LEFT TRANSVERSE AND SIGMOID SINUS THROMBOSIS



MRV
LEFT TRANSVERSE AND SIGMOID SINUS THROMBOSIS

Observation and Results

OBSERVATION AND RESULTS

Analysis of 50 female patients presenting with clinical symptoms suspicious of Cerebral Venous Thrombosis, admitted at Coimbatore Medical College and Hospital, between January 2006 to June 2007, who met the inclusion criteria was done.

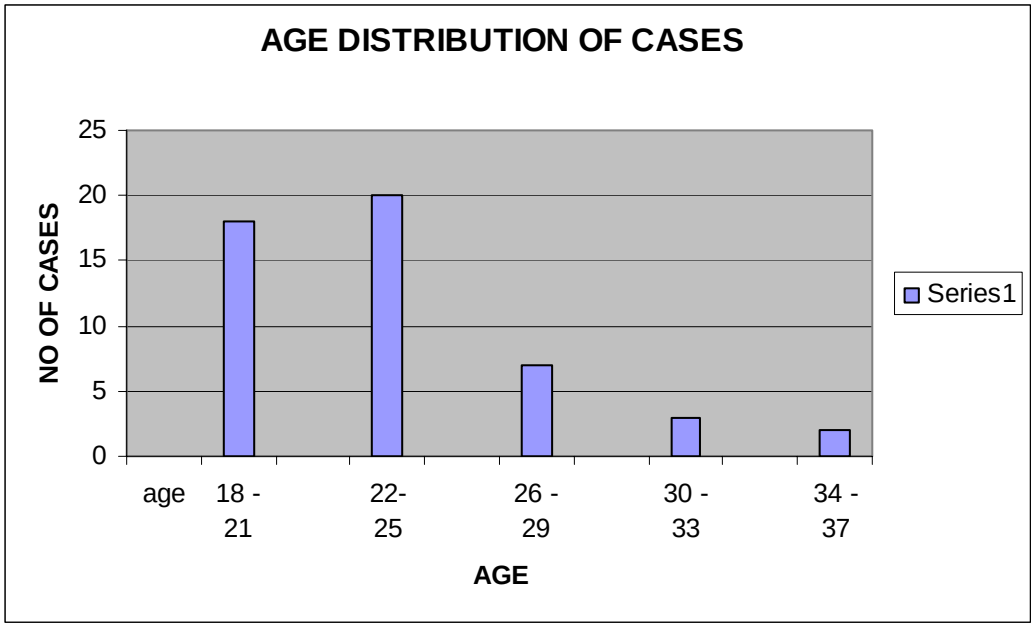
AGE WISE DISTRIBUTION OF CASES

Total cases - 50

AGE GROUP (yrs)	NUMBER OF CASES	PERCENTAGE
18 - 21	18	36
22 - 25	20	40
26 - 29	7	14
30 - 33	3	6
34 - 37	2	4

76% of patients presented in the age group between 18 – 25 years. Average age of patients presenting in the study is 24 years with a Standard Deviation of 4.05.

Applying the **Students T test T value = - 2.63 with a "p value of < 0.01 which is statistically significant.**

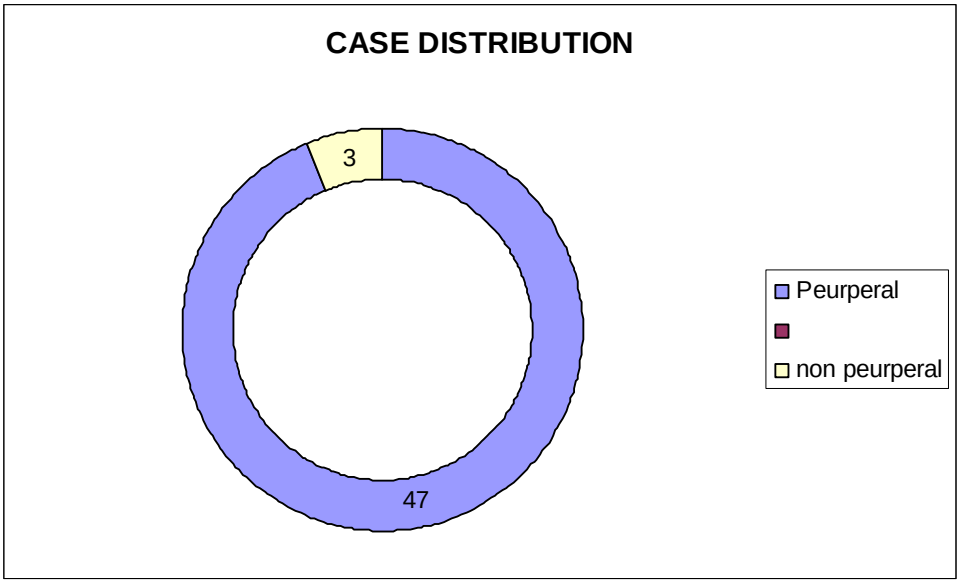


CASE DISTRIBUTION

	NUMBER	PERCENTAGE
PEURPERAL	47	94
NON PEURPERAL	3	6

Out of 50 patients analysed 47 cases were puerperal and 3 were non puerperal .

Applying the **Students T test , T = 1.49 and p = 0.06 , which is close to statistically significant**

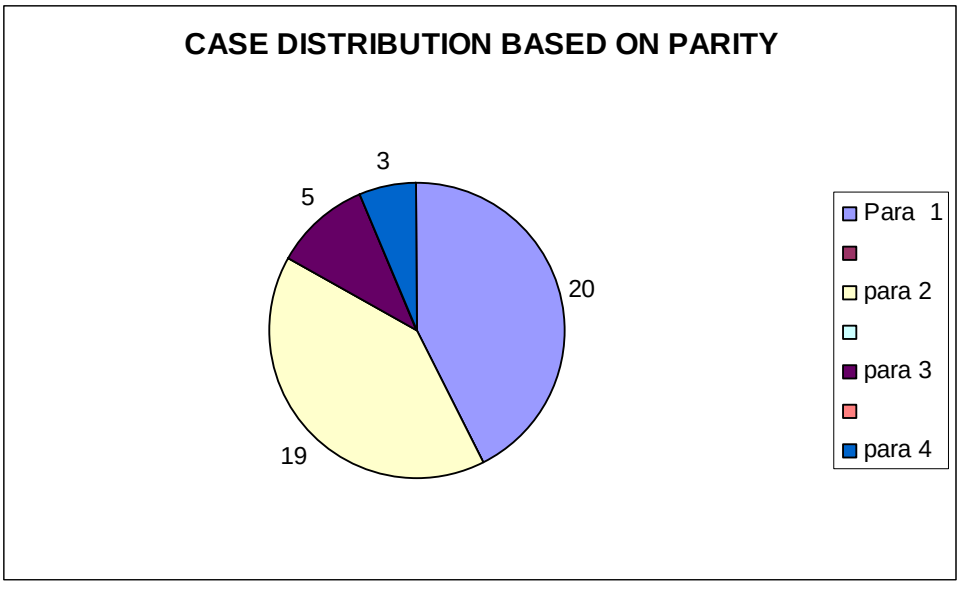


CASE DISTRIBUTION BASED ON PARITY

IN PEURPERAL CVT

PARITY	NUMBER	PERCENTAGE
PARA 1	20	42.6
PARA 2	19	40.4
PARA 3	5	10.6
PARA 4	3	6.4

On analyzing the case distribution of puerperal cases of CVT based on parity it was found that, it was more prevalent among multipara , 57.5% of cases . Among the multiparous it is more common in second parous . None of the patients were antenatal at presentation



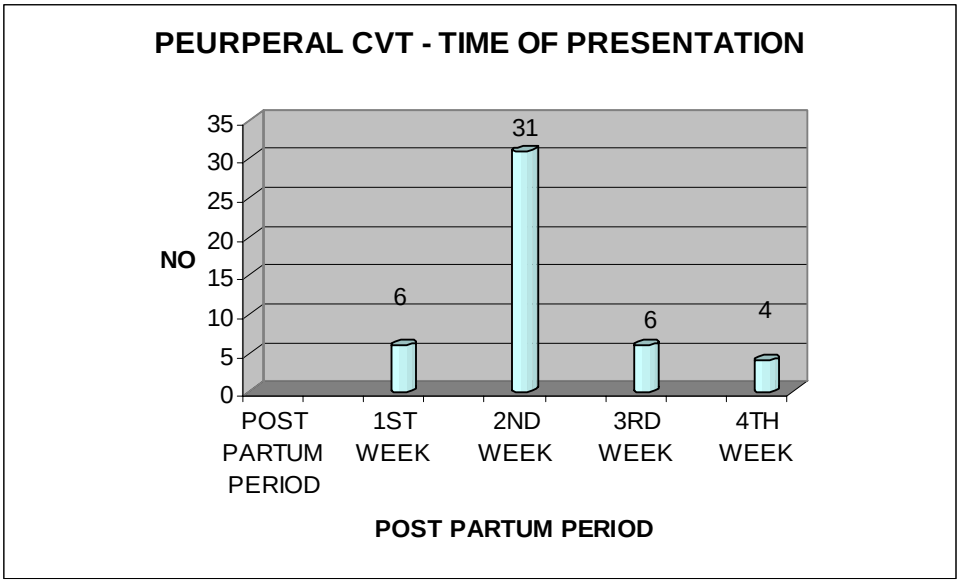
PEURPERAL CVT : TIME OF PRESENTATION

Total cases - 47

POST PARTUM PERIOD	NUMBER	PERCENTAGE
1ST WEEK	6	12.8
2ND WEEK	31	66
3RD WEEK	6	12.8
4TH WEEK	4	8.4

On analyzing the time of presentation in the puerperal cases it was found that 66% of cases presented in 2nd week .

On applying the **Students T test , T = 4.63 with a p value of < 0.01 which is statistically significant**



CLINICAL MODES OF PRESENTATION

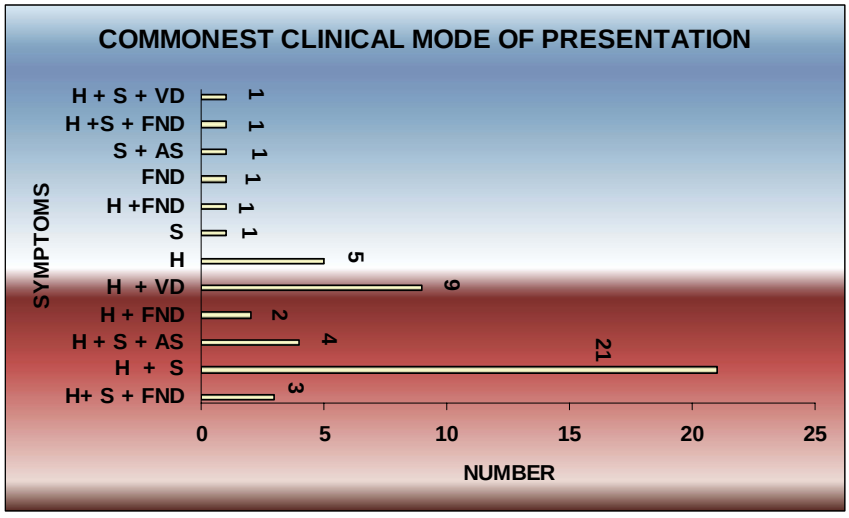
PRESENTING SYMPTOMS	NUMBER	PERCENTAGE
Headache (H) + seizures (S)	21	42
Headache (H) + seizures (S) +altered sensorium (AS)	4	8
Headache (H) + Focal neurological deficits (FND)	2	4
Headache (H) + seizures (S) +focal neurological deficits (FND)	3	6
Headache (H) + visual disturbances (VD)	9	18
Headache (H)	5	10
Seizures (S)	1	2
Headache (H) + Focal deficits (FND) + visual disturbances (VD)	1	2
Headache (H) + seizures (S) + visual disturbances (VD)	1	2
Focal deficits (FND)	1	2
Seizures (S) + altered sensorium (AS)	1	2
Headache (H) + seizures (S)+altered sensorium(AS) +focal deficits (FND)	1	2

On analyzing the commonest clinical mode of presentation it was found that headache was the universal symptom in 47 cases (94 %). The headache occurred commonly in association with other symptoms like seizures, visual disturbances, focal deficits and altered sensorium. In 5 (10%) it occurred in isolation. On applying the **Student T test, T value = - 0. 57 which is statistically significant.**

Of the cases presenting with seizures, 32 cases, 23 had generalized seizures and7 had focal seizures. The ten cases presenting with visual disturbances presented with visual blurring.

All the 8 cases of focal deficits presented in the form of hemiparesis.

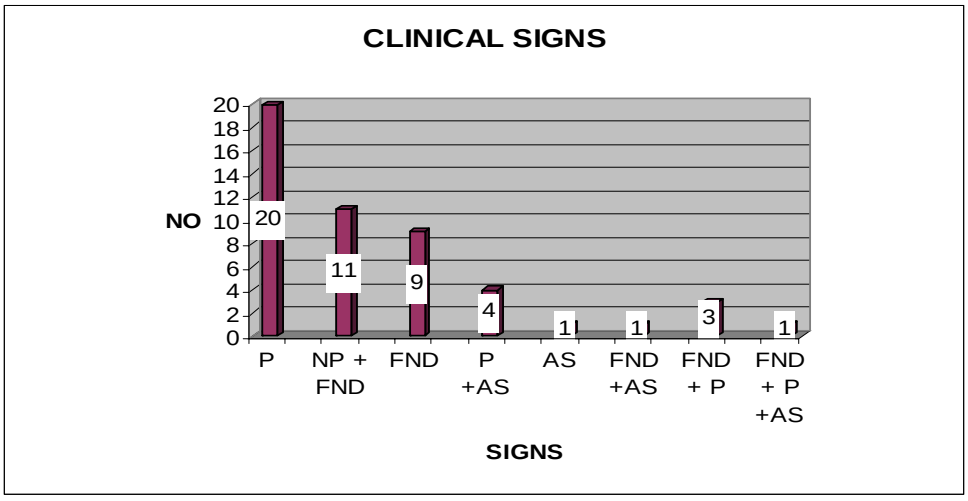
None of the cases presented with quadriparesis or paraparesis.



CLINICAL SIGNS

SIGNS	NUMBER	PERCENTAGE
PAPILLEDEMA(P)	20	40
FOCAL DEFICITS(FND) (hemiparesis)	9	18
PAPILLEDEMA + ALTERED SENSORIUM (stupor) (AS)	4	8
ALTERED SENSORIUM (coma)	1	2
FND + AS (coma)	1	2
FND + P	3	6
FND + P + AS	1	2
NO SIGNS	11	22

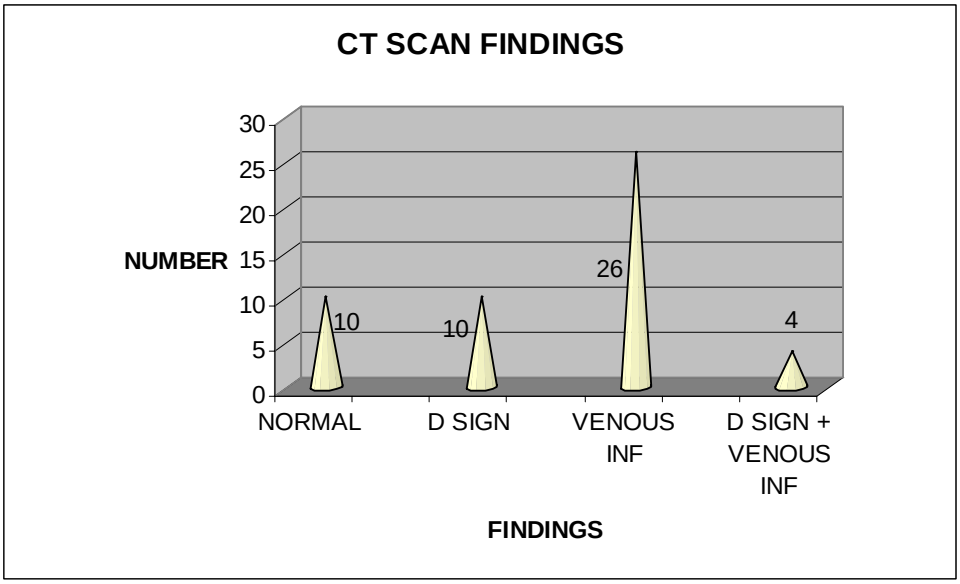
On analysis of the clinical signs it was found that 28 cases, 56% , had papilloedema, of which 20 had only papilloedema,4had papilloedema associated with altered sensorium, and 3 had associated hemiparesis. Of the 14 cases presenting with hemiparesis, 3 cases had facial weakness (7^{th} nerve palsy). No other cranial abnormality was detected.



C T FINDINGS

FINDINGS	NUMBER	PERCENTAGE
NORMAL	10	20
DELTA SIGN	10	20
VENOUS INFARCT	26	52
DELTA SIGN + VENOUS INFARCT	4	8

On analyzing the CT findings, it was found that 26 cases, 52%, had venous infarcts and all the infarcts were hemorrhagic. On applying the **Students T test – T= 0.3**, statistically significant. Nearly 10 cases, had normal CT.



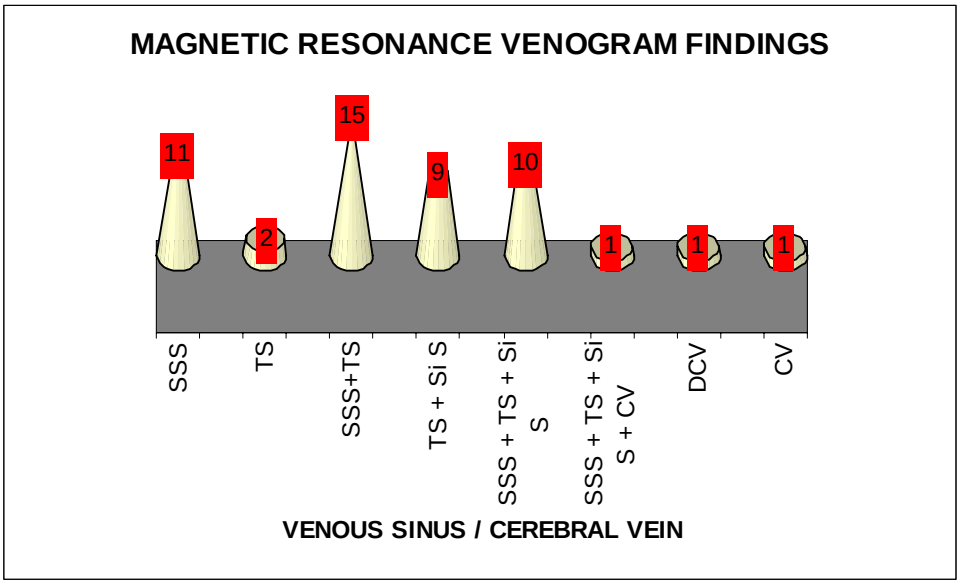
FINDINGS ON MAGNETIC RESONANCE VENOGRAM

VENOUS SINUS / CEREBRAL VEIN	NUMBER	PERCENTAGE
Superior Sagittal Sinus (S S S)	11	22
Transverse Sinus (T S)	2	4
S S S + T S	15	30
Transverse Sinus (T S) + Sigmoid sinus (Si S)	9	18
S S S + TS + Si S	10	20
S S S + TS + Si S + Cortical veins (CV)	1	2
Deep Cerebral Vein (DCV)	1	2
C V	1	2

On analyzing the topography of involved venous sinuses on Magnetic Resonance Venogram it was found that 37 cases, 74 %, had involvement of Superior Sagittal Sinus.

37 cases had Transverse sinus involvement, 2 cases in isolation and the rest 35 cases in association with involvement of other venous sinuses. parenchymal changes in the form of haemorrhagic venous infarcts, edema, minimal haemorrhages were noticed in 16 cases. None of the cases had involvement of cavernous sinus. 3 cases had extension of

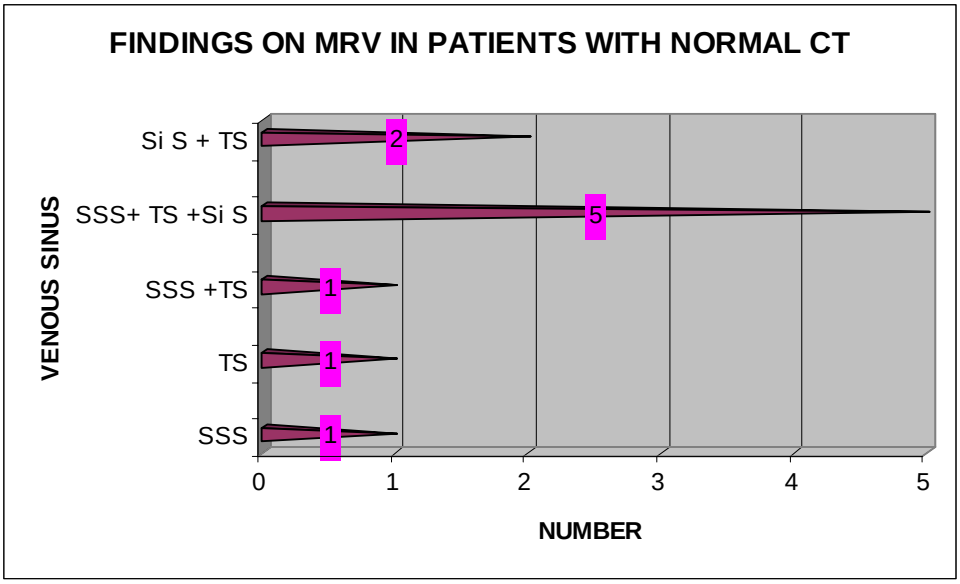
thrombus into internal jugular vein, 1 case in to straight sinus and vein of Galen.



M R V FINDINGS IN PATIENTS WITH NORMAL CT

VENOUS SINUS/ CEREBRAL VEIN	NUMBER	PERCENTAGE
S S S	1	10
T S	1	10
S S S + T S	1	10
S S S+ T S + Si S	5	50
Si S + T S	2	20

Of the 10 cases with normal CT scan , all the 10 had findings in MRV, 50% of which had involvement of multiple venous channels.

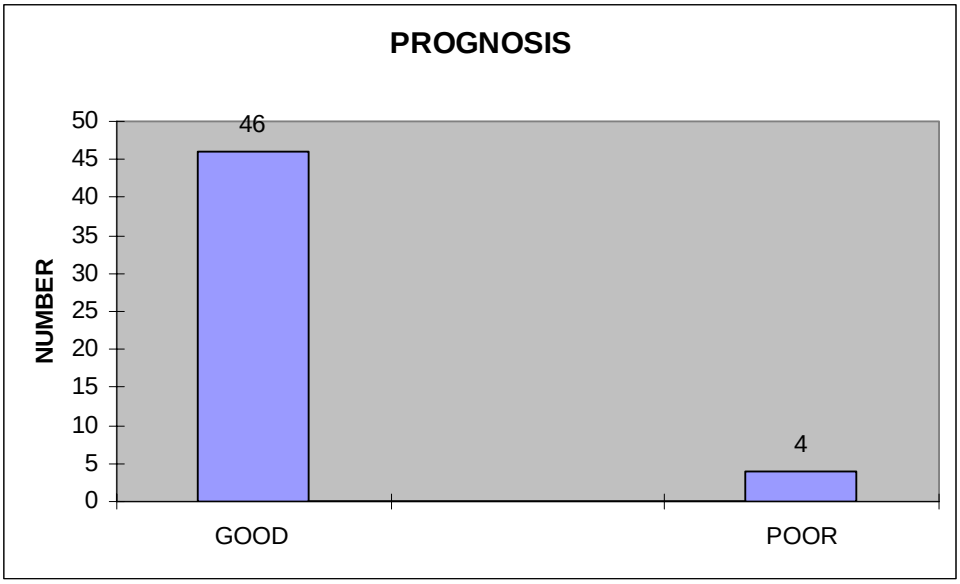


PROGNOSIS

	NUMBER	PERCENTAGE
GOOD	46	92
POOR	4	8

On analyzing the prognosis of patients at discharge it was found that 92 % had good prognosis and 8% had poor prognosis (death). Applying **Students T test, T= 0.78,** which is statistically significant.

Of the 4 cases, 3 cases had presenting symptoms of headache, seizures and altered sensorium and one case had headache, seizures and focal deficits. 2 cases had involvement of S S S, one case SSS + TS, and one case of deep cerebral vein.



OBSERVATION BASED ON LAB PARAMETERS :

On analyzing the lab parameters, anaemia was found in 15 cases, 30 % , of which 14 cases were in peurperium.

Peripheral smear in all 15 cases showed microcytic hypochromic picture

None of the patients had thrombocytosis or leukocytosis

Metabolic parameters like urea , creatinine and LFT and sugar values were normal

Coagulation profile – PT and APTT were normal

CSF analysis done in 22 cases were normal

ELISA for HIV and VDRL testing done in all cases were negative

COMPARISON WITH OTHER STUDIES

IN RELATION TO AGE

AMERI ET AL , 1992	AVG AGE – 20 - 30 YRS
PRESENT STUDY	AVG AGE - 18 – 25 YRS

IN RELATION TO CLINICAL SIGNS/ SYMPTOMS

SYMPTOMS	DUTCH – EUROPEAN STUDY 2001	MEHTA / MUTHU-KRISHNAN 2004	PRESENT STUDY
HEADACHE	95%	90%	94%
SEIZURES	47%	50%	64%
PARESIS	43%	9%	28%
PAPILLEDEMA	41%	78%	56%
ALTERED CONSCIOUSNESS	39%	-	14 %
RAISED INTRACRANIAL TENSION	22%	-	18%

IN RELATION TO MORTALITY

PROGNOSIS	WASAY ET AL 2001	PRESENT STUDY
GOOD	92	92
POOR / DEATH	8	8

Discussion

DISCUSSION

Cerebral venous thrombosis though not rare, is often undiagnosed as it presents with a wide array of symptoms .It mimics practically all neurological conditions. It affects all age groups and has unpredictable outcome. This condition is common in the Indian subcontinent, responsible for 10-15% of young strokes .It has been reported more commonly in women. Studies by various Indian authors (Agarwal1978, Srinivasan1983, Banerjee1973, Nagarajan 1988) have highlighted higher prevalence in pregnancy and puerperium.

In the present study **94%** of cases were puerperal ($p<0.06$-statistically significant). Though no uniform age group has been reported in many studies, most cases of CVT in women were reported in the age group of 20-35years,commonly related to pregnancy & puerperium and oral contraceptive use .In the present study mean age of presentation was **24 years** ($p<0.01$-stastistically significant).

Low socio-economic status, poor antenatal care, lack of iron fortification , fluid restriction and blood loss during pregnancy over and above the physiological changes ,have been proposed by various authors as the contributory cause for the higher incidence of CVT in our country.

These multifactorial causes could be cause for the higher prevalence of CVT in pregnancy and puerperium in the present study .None of our patients had any history suggesting thrombotic tendencies.

Present study has revealed a higher prevalence of CVT around **58%** in multiparous in accordance with various Indian studies (ChopraJS, BanerjeeAK 1989). **66%** of cases presented in the second week (8-14days) (p-0.01-stastically significant).

Extreme diversity in clinical presentation makes it a challenge to diagnose CVT on clinical grounds. In the present study headache was found to be the universal symptom in **94%** of cases (T-0.57-stastically significant), this is in accordance with various studies (Dutch-European2001; Mehta, Muthukrishnan2004). Headache was invariably accompanied by seizures 64%, focal deficits 16% and altered sensorium 12%.Thus headache presenting as the only sign of CVT is comparatively rare, in accordance with other studies (Deker, Steiner 2000; Saneto, Kinkel 2000).

Papilledema was detected to be the commonest sign occurring in **56%** cases followed by focal deficits in **28%** cases. The focal deficits

was in the form of hemi paresis, no cases presented paraparesis or quadriparesis. **26%** of the hemiparesis was left sided, the cause of this predominance is not known.

The diagnosis of CVT often requires neuroimaging. In the present study on cranial CT **52%** had venous infarcts (T-0.3-ststistically significant). All the venous infarcts were hemorrhagic. The classical delta sign was seen in 20% cases. CT was detected to be normal in 20% cases, in accordance with other studies (Ameri 1992; Bousser MG 2000).

The use of Magnetic resonance venogram has revolutionized the diagnosis of CVT .It has lead to rapid, noninvasive diagnosis of CVT and now is considered the gold standard .In the present study Superior sagittal sinus was detected to be the commonest venous channel involved in **74%** cases, 22% in isolation and 52% in association with other venous channels, this is in accordance with various studies (Dormont1994; kalbag1967; Mehta, Muthukrishnan2004). This manifested as raised intracranial tension. In the 20% cases that had normal cranial CT, MRV detected changes and venous sinus involvement in all the cases .Thus 20% of cases could escape detection and treatment delayed if CT alone is used as the only neuroimaging.

Anemia (Hb<12g%) was detected in 30% of cases, of which 28%were puerperal. Anemia has been recognized in most obstetric series of CVT (Bansal BC, Gupta RR, Srinivasan-1983), this deserves further studies to clarify its role in pathogenesis of CVT.

The role of Heparin in the management of CVT has been a matter of controversy. It's effectiveness, even in hemorrhagic venous infarcts has been extensively studied and recommended by various authors (Einhaupl 1991, Cepri 1998, Nagarajan 1998).Though most series have used high dose heparin as a continuous infusion in management of CVT ,limiting the use of low dose heparin(5000u s.c 8^{th} hourly) to puerperal cases ,in the present study all 50 cases were treated with unfractionated Heparin 5000 i.v 8^{th} hourly. All patients received oral anticoagulant for 3months.No complications were detected as a result of treatment regimen employed in the present study and proved to be effective.

Mortality in the present study is **8%**, correlating with that of literature (11%). Most of these cases, presented with raised intracranial tension, altered level of consciousness ,hemorrhagic venous infarcts and involvement of superior sagittal thrombosis on MRV. Thus presentation with altered level of consciousness could portend poor prognosis.

Thus CVT should be strongly considered in all cases presenting with a neurological problem, especially in the setting of pregnancy and puerperium. Neuroimaging with magnetic resonance venogram and treatment with heparin and search for the underlying cause is strongly recommended.

Conclusion

CONCLUSION OF THE STUDY

Cortical venous thrombosis was common in the age group of 22-25 years, with an average age of presentation at 24 years. It is more common among pregnancy and puerperium. Among puerperal cases it was common among multiparous, most cases were second para. Most cases of puerperal CVT presented in the second week.

Headache was the universal symptom, and usually occurred in association with other symptoms like seizures, visual disturbances and focal deficits. Papilledema was the commonest sign.

Majority of patients had hemorrhagic venous infarct on cranial CT. It was normal in 10 cases. 20% of patients with normal cranial CT had changes in the magnetic resonance venogram. Thus MRV is of great diagnostic utility and these 20% cases could escape detection if CT alone is used as the only neuro imaging modality. Commonest venous sinus involved in MRV was superior sagittal sinus and transverse sinus. Mortality was low (8%) and most cases had excellent recovery.

Bibliography

BIBLIOGRAPHY

1. Ribes MF. FRENCH Medical Journal 1825; 3:5

2. Aberocombei J. Superior sagittal thrombosis in puerperium 83. In pathological and practical Researches of the Brain and Spinal Cord.Edinburgh 1828 ; 83-85 cited by Donaldson JO ed; Neurology of pregnancy, Philadelphia,1978.

3. Kalbagh RM,Woif AL. Etiology of cerebral venous thrombosis in cerebral venous thrombosis: Oxford University Press, London 1967; pp238.

4. Bianchi D, Maeder P, Bogousslavesky J, et al. Diagnosis of CVT with routine magnetic resonance, an update. Eur Neurol 1998; 40(4):79-90

5. Hulcelle PJ, Dooms GC, Mathurin P, Cornelis G. MRI assessment of unsuspected dural sinus thrombosis. Neuroradiology. 1989;31:217-221.

6. Sze G, Simmons B, Krol G, Walker R, Zimmerman RD, Deck MDF. Dural sinus thrombosis: verification with spine echo technics. AJNR Am J Neuroradiol. 1988; 9: 679-686.

7. Nagaraja D, Tally AB. Stroke in the young in progress in clinical Neurosciences. Edt.publ Sinha KK, Ranchi 1988;2: 129-45.

8. Chopra JS, Prabhakar S. Clinical features and risk factors in stroke in young. Neurl Scandivia 1979; 43: 289-300.

9. Abraham J, Shetty C, Joss C. Stroke in the young. Stroke 1971; 2: 258-66.

10. Bansal BC, Gupta RR, Prakash C. Stroke during pregnancy and puerperium in young females below the age of 40 as a result of cerebral venous/venous sinus thrombosis. Jpn Heart J. 1980;21: 171-183.

11. Srinivasan K. puerperal cerebral venous arterial thrombosis. Semin Neural 1988; 8: 222-225.

12. Chopra JS, Banerjee AK. Primary intracranial sinovenous occlusions in youth and pregnancy. In: Vinken PJ, Bruyn GW, Klawans HL, eds. Handbook of Clinical Neurology, vol 10: Vascular Diseases, part II. New York/Amsterdam: Elsevier Science Publishing Co. Inc; 1989: 425-452.

13. Srinivasan K. Stroke in the young.Neurology in India 1988; 36: 189-94.

14. Srinivasan K.Cerebral venous and arterial thrombosis in pregnancy and peurperium.Angiology 1983;134: 737-46.

15. De Bruijin SF, Stam J, Koopman MM, Vandenbroucke JP. Case control study of risk of Cerebral sinus Thrombosis in OCP users and in carriers of hereditary prothrombolic condition. The CVT study group. BMJ 1998; 316; 589-92.

16. Bousser M.G; Cerebral venous thrombosis,diagnosis and management. J Neural.2000; 247: 252-58.

17. Peter M.Tzourio C, Ameri A, Bousser MG; Long- term prognosis in CVT, Follow up of 77 patients; Stroke 1996; 27: 243-246.

18. Stolz E, Kemkes-MMatthes B, Hahn M, et al;Screening for thrombophilic risk factors among 25 German patients with CVT. Acta Neural Scand 2000; 102: 31-36.

19. Bousser MG, Chiras J, Sauron B, Castaigra P. Cerebral venous thrombosis-review of 38 cases. Stroke 1985; 16: 199-213.

20. Carhuapoma JR, Mitsias P, Levine R.S; Cerebral venous thrombosis and anticardiolipin antibodies, Stroke 1997; 28: 2363-2369.

21. Bousser MG; CVT, Nothing, heparin or local thrombolysis ? Stroke 1999;30:481-483.

22. de Bruin S, de Haan R, Stam J. Clinical features and prognostic factors of CVT in a prospective series of 59 patients. J Neurol Neurosurg Psychiat 2001; 70: 105-8.

23. Barnett HJM, Hyland HH; Noninfective intracranial venous thrombosis.Brain 1953; 76: 36-45

24. Ameri A, Bousser MG.Cerebral venous sinus thrombosis. Neurol Clin 1992; 10:87-111.

25. Roos KL, Pescuzzi RM, kuharik MA, Shapiro AD, Manco-Johnson MJ. Postpartum intracranial venous thrombosis associated with dysfunctional protein C and deficiency of protein S. Obstet Gynecol 1990; 76:492-4.

26. Martin J, Sheehan H.Primary thrombosis of cerebral veins (following childbirth). Br.Med J 1941; 1:349-53.

27. Canu C, Barinagarrementeria F. CVT associated with pregnancy and puerperium: a review of 67 cases. Stroke 1993; 305:28-35.

28. Huges CRV. The antiphospholipid syndrome. Lancet 1993; 342: 341-4.

29. Deschiens M, Conard J, Horellou M, Ameri A, Preter M, Chedu F. Coagulation studies, factor V Leiden and anticardiolipin antibodies

in 40 cases of Cerebral venous sinus thrombosis. Stroke 1996; 27: 1724- 27:1724-30.

30. Pasquale LR, Moster ML, Schmaier A. Dural sinus thrombosis with abnormalities of protein S and fibrinogen. Arch Ophthalmol 1990; 108: 644.

31. HauserD, Barzilai N, Zalish M, Pollack A. Bilateral pailloedema with retinal hemorrhages in association with cerebral venous sinus thrombosis and paroxysmal nocturnal hemoglobinuria. Am J Ophthalmol 1996; 122: 592-3.

32. Feldenzer JA, Bueche MJ, Venes JL, Gebarski SS. Superior sagittal sinus thrombosis with infarction in sickle cell trait. Stroke 1987;; 18:656-60.

33. Schwab FJ, Peyster RG, Brill CB. CT of cerebral venous sinus thrombosis in a child with homocystinuria.paediatric radiol 1987;17;244-5.

34. Akatsu H, Vaysburd M, Fervenza F, Peterson J, Jacobs M. Cerebral venous thrombosis in nephrotic syndrome. Clin Nephrol 1997; 48: 317-20.

35. David RB, Hadfield MG, Vines FS, Maurer HM, Dural sinus occlusion in leukemia .Peadiatrics 1975, 56:793-6.

36. Ellie E, Beoran Y, Brunet R. Paraneoplastic cerebral venous thrombosis. France Review Neurology 1993; 149: 297-9.

37. Reddingius RE, Patte C, Couanet D, kalifa C, Lemerle J. dural sinus thrombosis in children with cancer. Med Pediatr Oncol 1997; 29: 296- 302.

38. Sigsbee B; Deck MD; Posner JB. Non metastatic superior sagittal sinus thrombosis complicating systemic cancer. Neurology 1979; 29:139-46.

39. Ohata K; Hque M, Morino M, Nagai K, Nisho A, Nishijima Y, Hakubs A. Occlusion of the sigmoid sinus after surgery via the presigmoidal-transpetrosal approach. J Neurosurg 1998; 89: 575-84.

40. Saneto P.R,Samples S, Kinkel P. Traumatic intracerebral venus thrombosis associated with an abnormal golf swing, Headache 2000: 40 595-598.

41. Wassermann D. Acute paranasal sinusitis and cavernous sinus thrombosis. Arch Otolaryngol 1967; 86: 205-9.

42. Grafstein E, Fernandes CM, Samoyloff S. Lateral sinus thrombosis complicating mastoiditis. Ann Emerg Med 1995; 25:420-3.

43. Murthy JMK, Chopra JS, Malik SR et al.Clinico-pathological correlates in autopsy in pyogenic meningitis.Ind J Med Res 1883; 78: 234-52.

44. Mathew NT, Myers JS. Taori CM et al. Internal carotid artery thrombosis in cavernous sinus thrombosis. Arch Neuro 1971; 24: 11-5.

45. Kelly MA, Gorelick PB, Mirza D. The role of drugs in the etiology of stroke. Clin Neuropharmacol 1992; 15: 249- 75.

46. Buchanan DS, Brazinsky JH. Dural sinus and cerebral venus thrombosis: incidence in young women receiving oral contraceptives. Arch Neurol 1970;22:440-4.

47. Srachan R, Hughes D, Cowie R. Thrombosis of the straight sinus complicating hormone replacement therapy. Br.J Neurosurg 1995; 9:805-8.

48. Jaillard AS, Hommel M. Venous sinus thrombosis associated with androgens in a healthy young man. Stroke 1994; 25:212-13.

49. Rothwell PM, Grant R. Cerebral venous sinus thrombosis induced by ‘ ecstasy’. J Neurol Neurosurg Psychiatry 1993;56:1035.

50. Meyer-Lindenberg A, Quenzel EM, Bierhoff E, Wolf h, Schindler E, Biniek R. Fatal cerebral venous sinus thrombosis in heparin induced thrombotic thrombocytopenia. Eur Neurol 1997;37:191-2.

51. Musio F, Older SA, Jenkins T, Gregorie em. Case report: CVT as a manifestation of acute ulcerative colitis. Am J Med Sci 1993;24:1880-4.

52. Byrne JV, Lawton CA. Meningeal sarcoidosis causing intracranial hypertension secondary to dural sinus thrombosis. Br. J.Rdiol 1983;56:755-7.

53. el-Ramahi KM, al-Kawi MZ.Papiloedema in Bechcet’s disease; value of MRI in diagnosis of dural sinus thrombosis. J Neurol Neurosurg Psychiatry 1991; 54:826-9.

54. Shiozawa Z, Yoshida M, Kobayashi K, Tsunoda S, Mano T. Superior sagittal sinus thrombosis and systemic lupus erythematosus. Ann Neurol 1986;20:272.

55. Urban E, Jabbari B, Robles H.Concurrent cerebral venous sinus thrombosis and myeloradiculopathy in Sjogren's syndrome. Neurology 1994;44:554-6.

56. Srinivasan K. Puerperal CVT Seminar in Neurology 1988;8:222-5.

57. Dalal PM. Stroke in India. Jap Heart Jour 1982;46:621-5.

58. Daif A, Awada A, Al-Rajeh et al.Malibary CVT in adults a study of 40 cases from Saudi Arabia. Stroke 1995;26:1193-5.

59. Prakash C, Singh S. CVT in puerperium. J Ass Phys India 1960;8:363-5.

60. Srinivasan K, Ramamurthy B. Neurological disorder in pregnancy and puerperium. J Ass Phy India 1971; 19:705-07.

61. Janaki S, Thomas L. Neurological complications in pregnancy and puerperium. Neurology India 1963;11:128-30.

62. Dibakdsib JO. Cerebrovascular disease. In: Donaldson JO. Ed. Neurology of pregnancy. Philedelphia, Pa: WB Saunders: 1978: 135-34.

63. Lanska DJ, Kryscio RJ. Risk factors for peripartum and postpartum stroke and intracranial venous thrombosis. Stroke 2000; 31:1274-82.

64. Cumming AM, Tait RC, Fildes S, Yoong A, Keeny S, Hay CRM. Development of resistance to activated protein C during pregnancy. Br. J Haematol 1995;90:725-7.

65. Vandenbroucke J. Cerebral sinus thrombosis and oral contraceptive(editorial). BMJ1998;317:878-9.

66. Prakash C,Arya RK, Singla KP et al.Platelet adhesiveness and serum lipids in CVT. J Ass Phy Ind 1970;18:816-9.

67. Bansal BC, Prakash C, Gupta RR et al. Study of serum lipids and fibrinolytic activity in CVT during puerperium. Am J Obst Gyn 1974;119(8):1079-82.

68. Bansal BC, Prakash C.Serum lipids, platelet and fibrinolytic activity in cerebrovascular disease. Stroke 1978;9:137-9.

69. de Bruijjb S, Stam J, Kaappelle L. Thunderclap headache as the first symptom of cerebral venus sinus thrombosis. Lancet 1996; 348:1623-5.

70. Deker R.R, Steiner I. Isolated intracranial hypertension as the only sign of cerebral venous thrombosis. Neurology 2000;54:2030.

71. Jacobs K, Moulin T, Bougousslavsky J, Woimant F, Dehaene I, Tatu L, Besson G, Assouline E, Casselman J. The stroke syndrome of cortical vein thrombosis. Neurology 1996;47:376-82.

72. Hayley EC, Brashear HR, Barth JT, Cail WS, Kassell N. Deep cerebral venous thrombosis: clinical, neuroradiological and neuropsychological correlates. Arch Neurol 1989; 46:337-40.

73. Nayak AK, Karnad D, Mahajan MV, Shah A, Meisheri YV. Cerebellar venous infarction in chronic suppurative otits media: a case report with review of four other cases. Stroke 1994;25:1058-60.

74. Banerjee AK, Chopra JS, Sawhney BB. Puerperal cerebral venous thrombosis. Study of autopsy material. Neurology Journal of Indian Academy of Clinical Medicine.vol.5 No.1 61 India 1973;21:19-22.

75. Tsai FY, Wang AM, Matovich VB, Lavin M, Berberian B, Simonson tm, Yuh WT.MR staging of acute dural sinus thrombosis: correlation with venous pressure measurements and implications for treatment and prognosis. Am J Neuroradiol 1995;16:1021-9.

76. Diaz JM, Schiffman JS, Urban ES, Maccario M. Suprior sagittal sinus thrombosis and pulmonary embolism: a syndrome rediscovered. Acta neurol Scand 1992;86:390-6.

77. Hladky JP, Leys D, Vantyghem MC, Furby A, Leclerle X, Dupard T, Lefebvre J. Early hypopituitarism following cavernous sinus thrombosis: total recovery within 1 year. Clin Neurol Neurosurg 1991;93:249-52.

78. Houser OW, Campbell JK, Campbell RJ, Sundt TM. Arteriovenous malformation affecting the transverse dural venous sinus: an acquired lesion. Mayo Clin Proc 1979;54:651-61.

79. Brant-Zawadzki M, Chang ZY, Meccarty GE. Computed tomography in dural sinus thrombosis. Arch Neuro 1982.

80. Maheswari MC, Bhargave S. Evaluation of peripartum stroke by C.T. Scan. Clinical Radiology 1983;34:633-7.

81. Rao KCVG; Knipp HC, Wagner EJ, CT Findings in cerebral sinus and venous thrombosis. Radiology 1981; 140:391-98.

82. Bousser MG; Ross Russeu R. CVT vol.1, London: WB Saunders 1997; 175.

83. Patronas NJ, Duda EE, Mifakhrace M, Wolmann RL. Superior sagittal sinus thrombosis. Diagnosed by computed tomography. Surg Neurol 1981;15:11-14.

84. Bouonanna F, Moody DM, Ball MR, Laster DW. Computer cranial tomographic findings in sino-venous occlusion. J Computer Assisted tomographic 1978;2:281-90.

85. Dormont D, Axionnat R et al. MRI in cerebral venous thrombosis. J Neuroradiology 1994;21(2):81- 99.

86. Vogl T. Bergman C, Villringer A, Einhaupal K, Lissner J, Felix R. Dural sinus thrombosis:value of venous MRA for diagnosis and follow up. Am J Radiology 1994;162:1191-8.

87. Padayachee TS, Bingham JB, Grave MJ. Dural sinus thrombosis. Diagnosis and follow up by magnetic resonance, angiography and imaging. Neurology 1991;33:165-7.

88. Provenzale JM, Joseph GJ, Barboriak DP. Dural sinus thrombosis; findings on CT and MR imaging and diagnostic pitfalls. Am J Radiology 1998;170:777-83.

89. Barnes BD, Winestock DP. Dynamic radionuclide scanning in the diagnosis of thrombosis of superior sagittal sinus. Neurology 1977;27:656.

90. Lalive PH, de Moerloose P, Lovblad K, et al: Is measurement of D-dimer useful in the diagnosis of CVT? Neurol 2003 Oct 28; 61(8):1057-60.

91. Einhaupl EM, Villringer A et al. Heparin in cerebral venous thrombosis. Labcet 1991;338:597-600.

92. Dirocco C,Lannelli A, Leonac G et al. Heparin-urokinase treatment in aseptic dural sinus thrombosis. Arch Neuro 1981;38:431-5.

93. Cepri S; Gangeni A, Campolo C et al. High dose heparin plus warfarin administration in non traumatic dural sinus thrombosis- a clinical and neurological study. J Neurosurgical Scie 1998;42(1): 23-32.

94. Nagaraja D,Tlly AB, Hardas VT et al. Heparin in haemorrhagic infarction in CVT. J Ass Phy Ind 1998;46:706-07.

95. Bruuckler MB, Volleret-Rogenhofer H, Wagner M, Steilbauer-Felber S, Trenkler J, Deisenhammer E, Aichner F. Heparin treatment in acute cerebral sinus thrombosis: a retrospective clinical and MR analysis of 42 cases. Cerebrovascular Dis 1998;8:331-3.

96. Einhaupl K, Villringer A, Meister W, Mehraein S, Garner C, Pellkofer M, Haberl R, Pfister HW, Schmiedek P. Heparin treatment in venous sinus thrombosis. Lancet 1991;338:597-600.

97. Levine SR, Twyman RE, Gilman S. The role of anticoagulation in cavernous sinus thrombosis. Neurology 1998 Apr; 38(4):517-22.

98. Scott JA, Pascuzzi RM, Hall PV, Becker GJ.Treatment of dural sinus thrombosis with local urokinase infusion. J Neurosurg 1988; 68:284-7.

99. Horowitz M, Purdy P, UNwin H, Carstens G, Greenlee R, Hise J, Kopitnik T, Batjer H, Rollins N,Samson D. Treatment of dural sinus thrombosis using selective catheterization and urokinase. Ann Neurol 1995;38:58-67.

100. Kim SY, Suh JH. Direct endovascular thrombolytic theraphy for dural sinus thrombosis: Infusion of alteplase. Am J Neuroradiol 1997; 18:639-45.

101. Frey JL, Muro GJ, Mcdougall CG, Dean BL, Jahnke HK. CVT: Combined intra thrombus rtPA and i.v heparin. Stroke 1999; 30:489-94.

102. Benamer HTS, Bone I. CVT: anticoagulants or thrombolytic therapy? J Neurol Neurosurg Psychiat 2000;69:427-30.

103. Derex L, Philppean F, Nighoghossian N et al. Postpartum CVT. J Neurol Neurosurgery 1988;65(5):80-2.

104. Nagpal RD,. Dural sinus and CVT. Neurosurg Rev 1983;6:155-60.

105. Zilkha A, Stenzler SA, Lin JH. Computed tomography of the normal and abnormal superior sagittal sinus. Clin Radiol 1982;33:415-25.

106. Preter M, Tzourio C, Ameri A, Bousser MG. Long-term prognosis in CVT: follow up of 77 patients. Stroke 1996;27:243-6.

107. de Bruuijn SF, Budde M, Teunisse S, de Haan RJ. Long term outcome of cognition and functional health after cerebral venous sinus thrombosis. Neurology 2000;54:1687-9.

108. Dormont D, Anxionnat R, Evrard S, Louaillle C, Chiras J, Marsault C. MRI in CVT. J neuroradiol 1994;21:81-99.

109. Krristensen B, Malm J, Markgren P, Ekstedt J. CSF hydrodynamics in superior sagittal sinus thrombosis. J Neurol Neurosurg Psychiat 1992;55:287-93.

Appendix

COIMBATORE MEDICAL COLLEGE AND HOSPITAL

PROFORMA

NAME : AGE: SEX: I.P NO:

D.O.A: D.O.D: NEURO NO:

COMPLAINTS:

HISTORY OF:

FEVER:
EAR DISCHARGE
DIARRHOEA
HEMORRHAGIC TENDENCIES
THROMBOTIC TENDENCIES
P.I.H
RECURRENT PREG LOSS
MALIGNANCIES
PROLONGED IMMOBILISATION
CHRONIC DRUG INTAKE

OBSTETRIC H/O: G P L.C.B

EXAMINATION

GENERAL PHYSICAL EXM:

PULSE: B.P TEMP RESP RATE

C.V.S :

R.S /E.NT :

PER ABDOMEN :

CENTRAL NERVOUS SYSTEM :

LAB DIAGNOSIS :

1. COMPLETE BLOOD COUNT :
2. PERIPHERAL SMEAR :
3. URINE ROUTINE :
4. BLOOD UREA :

   SUGAR :

   SREUM CREATININE :
5. LIVER FUNCTION TESTS :
6. PROTHROMBIN TIME :
7. a.P.T.T.:
8. VDRL/HIV:
9. C.S.F.ANALYSIS:
10. C.T. BRAIN-PLAIN/CONTRAST:
11. M.R.V:

PROGNOSIS :

Master Charts

S.No.	Age	Complaints							History of										Obstetric History			Examination								
		Headache	Seizures		Focal Deficits	Visual Disturbances	Altered Sensorium	Other Cranial Symptoms	Fever	Ear Discharge	Hemorrhagic Tendency	Thrombotic Tendency	RPL	PIH	Malignancies	Chronic Drug Intake	Diarrhoea / dehydration	Prolonged immobilisation	Gravida	Para	LCB	GPE	Pulse/mt	BP mm/hg	Temp ^{O}F	CVS	RS	Abd	CNS
			F	GTCS																									
1	23	P	A	P	A	A	P	A	A	A	A	A	A	A	A	A	A	A	-	P_1	15d	Anemia	N	N	N	N	N	N	Stupor L Hemiparesis L VII Palsy
2	25	P	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_2	8d	Anemia	N	N	N	N	N	N	B/L papilledema
3	25	A	A	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_2	3d	Anemia	N	N	N	N	N	N	L Hemiparesis
4	18	P	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_1	15d	Anemia	N	N	N	N	N	N	Normal
5	26	P	A	P	A	A	P	A	A	A	A	A	A	A	A	A	A	A	-	P_1	10d	N	N	N	N	N	N	N	Coma. Fundus N
6	20	P	A	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_1	10d	N	N	N	N	N	N	N	L Hemiparesis
7	23	P	A	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_1	30d	N	N	N	N	N	N	N	L Hemiparesis B/L papilledema
8	25	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_1	10d	N	N	N	N	N	N	N	Normal
9	24	P	A	A	A	P	A	A	A	A	A	A	A	A	A	A	A	A	-	P_1	10d	Anemia	N	N	N	N	N	N	B/L papilledema
10	18	P	A	P	A	A	P	A	A	A	A	A	A	A	A	A	A	A	-	P_1	8d	N	N	N	N	N	N	N	Stupor B/L papilledema
11	20	P	A	A	A	P	A	A	A	A	A	A	A	A	A	A	A	A	-	Nulli	-	Anemia	N	N	N	N	N	N	B/L papilledema
12	29	P	A	A	A	P	A	A	A	A	A	A	A	A	A	A	A	A	-	P_1	2yrs	N	N	N	N	N	N	N	B/L papilledema

S.No.	Age	Complaints							History of										Obstetric History			Examination								
		Headache	Seizures		Focal Deficits	Visual Disturbances	Altered Sensorium	Other Cranial Symptoms	Fever	Ear Discharge	Hemorrhagic Tendency	Thrombotic Tendency	RPL	PIH	Malignancies	Chronic Drug Intake	Diarrhoea / dehydration	Prolonged immobilisation	Gravida	Para	LCB	GPE	Pulse/mt	BP mm/hg	Temp ^{O}F	CVS	RS	Abd	CNS
			F	GTCS																									
13	22	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_2	9d	N	N	N	N	N	N	N	B/L papilledema
14	25	P	-	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_2	15d	Anemia	N	N	N	N	N	N	B/L papilledema, Stupor
15	27	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_1	3d	N	N	N	N	N	N	N	L Hemiparesis L VII Palsy
16	26	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_1	11d	Anemia	N	N	N	N	N	N	Normal
17	25	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_3	15d	N	N	N	N	N	N	N	B/L papilledema
18	21	P	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_2	10d	N	N	N	N	N	N	N	Normal
19	21	P	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_1	10d	Anemia	N	N	N	N	N	N	Normal
20	23	P	A	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_2	30d	N	N	N	N	N	N	N	L Hemiparesis L VII Palsy
21	21	P	P	A	A	A	P	A	A	A	A	A	A	A	A	A	A	A	-	P_1	22d	N	N	N	N	N	N	N	Coma. B/L papilledema

S.No.	Age	Complaints							History of										Obstetric History			Examination								
		Headache	Seizures		Focal Deficits	Visual Disturbances	Altered Sensorium	Other Cranial Symptoms	Fever	Ear Discharge	Hemorrhagic Tendency	Thrombotic Tendency	RPL	PIH	Malignancies	Chronic Drug Intake	Diarrhoea / dehydration	Prolonged immobilisation	Gravida	Para	LCB	GPE	Pulse/mt	BP mm/hg	Temp ^{O}F	CVS	RS	Abd	CNS
			F	GTCS																									
22	18	P	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_2	20d	N	N	N	N	N	N	N	B/L papilledema
23	34	P	P	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	-	Nulli	-	N	N	N	N	N	N	N	L Hemiparesis
24	22	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_2	15d	N	N	N	N	N	N	N	B/L papilledema
25	24	A	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_2	5d	Anemia	N	N	N	N	N	N	Normal
26	21	P	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_1	3d	N	N	N	N	N	N	N	B/L papilledema
27	20	P	A	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_1	10d	N	N	N	N	N	N	N	L Hemiparesis B/L papilledema
28	22	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_2	30d	Anemia	N	N	N	N	N	N	Normal

S.No.	Age	Complaints							History of										Obstetric History			Examination								
		Headache	Seizures		Focal Deficits	Visual Disturbances	Altered Sensorium	Other Cranial Symptoms	Fever	Ear Discharge	Hemorrhagic Tendency	Thrombotic Tendency	RPL	PIH	Malignancies	Chronic Drug Intake	Diarrhoea / dehydration	Prolonged immobilisation	Gravida	Para	LCB	GPE	Pulse/mt	BP mm/hg	Temp ^{O}F	CVS	RS	Abd	CNS
			F	GTCS																									
29	20	P	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_1	11d	N	N	N	N	N	N	N	Normal
30	23	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_2	14d	N	N	N	N	N	N	N	L Hemiparesis
31	26	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_2	10d	N	N	N	N	N	N	N	B/L papilledema
32	32	P	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_4	4d	N	N	N	N	N	N	N	B/L papilledema
33	20	P	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_2	10d	N	N	N	N	N	N	N	B/L papilledema
34	20	P	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_3	8d	N	N	N	N	N	N	N	L Hemiparesis
35	18	P	A	A	A	P	A	A	A	A	A	A	A	A	A	A	A	A	-	P_1	10d	N	N	N	N	N	N	N	B/L papilledema
36	22	P	A	A	P	P	A	A	A	A	A	A	A	A	A	A	A	A	-	P_2	12d	N	N	N	N	N	N	N	B/L papilledema R Hemiparesis
37	25	P	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_3	8d	Anemia	N	N	N	N	N	N	Normal

S.No.	Age	Complaints							History of										Obstetric History			Examination								
		Headache	Seizures		Focal Deficits	Visual Disturbances	Altered Sensorium	Other Cranial Symptoms	Fever	Ear Discharge	Hemorrhagic Tendency	Thrombotic Tendency	RPL	PIH	Malignancies	Chronic Drug Intake	Diarrhoea / dehydration	Prolonged immobilisation	Gravida	Para	LCB	GPE	Pulse/mt	BP mm/hg	Temp ^{O}F	CVS	RS	Abd	CNS
			F	GTCS																									
38	30	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_4	12	Anemia	N	N	N	N	N	N	B/L papilledema
39	19	P	A	P	A	P	A	A	A	A	A	A	A	A	A	A	A	A	-	P_1	10d	N	N	N	N	N	N	N	B/L papilledema
40	35	P	A	A	A	P	A	A	A	A	A	A	A	A	A	A	A	A	-	P_1	10d	N	N	N	N	N	N	N	B/L papilledema
41	20	P	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_2	10d	N	N	N	N	N	N	N	L Hemiparesis
42	22	P	A	A	A	P	A	A	A	A	A	A	A	A	A	A	A	A	-	P_2	13d	N	N	N	N	N	N	N	B/L papilledema
43	18	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_1	10d	N	N	N	N	N	N	N	Normal
44	25	A	A	p	A	A	P	A	A	A	A	A	A	A	A	A	A	A	-	P_1	10d	N	N	N	N	N	N	N	L Hemiparesis
45	27	P	A	A	A	P	A	A	A	A	A	A	A	A	A	A	A	A	-	P_3	14d	N	N	N	N	N	N	N	Stupor B/L papilledema

S.No.	Age	Complaints							History of										Obstetric History			Examination								
		Headache	Seizures		Focal Deficits	Visual Disturbances	Altered Sensorium	Other Cranial Symptoms	Fever	Ear Discharge	Hemorrhagic Tendency	Thrombotic Tendency	RPL	PIH	Malignancies	Chronic Drug Intake	Diarrhoea / dehydration	Prolonged immobilisation	Gravida	Para	LCB	GPE	Pulse/mt	BP mm/hg	Temp ^{O}F	CVS	RS	Abd	CNS
			F	GTCS																									
46	20	P	A	A	A	P	A	A	A	A	A	A	A	A	A	A	A	A	-	P_1	8d	N	N	N	N	N	N	N	B/L papilledema
47	22	P	A	P	P	A	P	A	A	A	A	A	A	A	A	A	A	A	-	P_2	10d	N	N	N	N	N	N	N	Stupor B/L papilledema L Hemiparesis
48	24	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_2	12d	N	N	N	N	N	N	N	B/L papilledema
49	30	P	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	-	P_4	14d	Anemia	N	N	N	N	N	N	Normal
50	28	P	A	A	A	P	A	A	A	A	A	A	A	A	A	A	A	A	-	P_3	7d	Anemia	N	N	N	N	N	N	B/L papilledema

S.No.	Complete Hemogram																		Magnetic Resonance Venogram						
	Hb g%	TC/cu mm	DC	ESR mm/hr	RBC mil/cu mm	Platelets lac/cu mm	Peripheral Smear	Urine	Blood Urea md/dl	Blood Sugar mg/dl	Serum Creatinine mg/dl	LFT	PT	aPTT	HIV	VDRL	CSF	Cranial CT Plain / Contrast	SSS	TS	SS	CV	DCV	Parenchyma	Prognosis
1	7.0	5200	P_{70} L_{30}	10	3.0	2.0	Microcytic Hypochromic	N	20	80	1.0	N	10	30	NEG	NR	N	Hemorrhagic Infarct R Frontal	+	+	-	-	-	Venous Infarct with Surrounding Edema R Frontal	G
2	7.6	4600	P_{60} L_{40}	15	5.0	1.5	Microcytic Hypochromic	N	22	86	1.0	N	15	29	NEG	NR	ND	Hemorrhagic Infarct B/L Frontal	+	+	+	-	-	-	G
3	6.9	5000	P_{70} L_{30}	14	4.2	2.0	Microcytic Hypochromic	N	21	100	0.8	N	14	27	NEG	NR	N	Hemorrhagic Infract L parieto occipital	-	+	+	-	-	-	G
4	8.0	4100	P_{66} L_{32} E_2	12	3.6	1.8	Microcytic Hypochromic	N	20	96	1.3	N	15	30	NEG	NR	N	Hemorrhagic Infarct R Frontal	+	-	-	-	-	-	G
5	12.0	5200	P_{70} L_{30}	10	30.0	2.0	N	N	24	98	0.8	N	15	28	NEG	NR	N	Hemorrhagic Infarct L Frontal	+	-	-	-	-	-	D
6	13.0	4000	P_{60} L_{40}	10	3.2	2.5	N	N	26	88	0.8	N	16	24	NEG	NR	N	Normal	+	+	+	-	-	-	G
7	12.0	6100	P_{75} L_{25}	13	4.0	2.0	N	N	20	84	0.9	N	15	26	NEG	NR	ND	Normal	+	+	-	-	-	Venous Infarct with Vasogenic Edema R Para Sagittal	G
8	12.0	5600	P_{60} L_{38} E_2	14	4.0	2.0	N	N	21	82	1.0	N	15	31	NEG	NR	N	Hemorrhagic Infarct L Parietal	-	+	+	-	-	Extension IJV + Venous Hemorrhagic Infract Parieto Temporo Occipital	G
9	7.0	5200	P_{70} L_{30}	10	3.5	2.0	Microcytic Hypochromic	N	20	90	1.0	N	15	30	NEG	NR	ND	Normal	-	+	+	-	-	-	G
10	12.0	5600	P_{70} L_{30}	12	4.0	1.8	N	N	18	94	1.5	N	11	32	NEG	NR	ND	Hemorrhagic Infarct L Temporo Occipital	-	+	+	-	-	Large Temporo Occipital Hematoma with subdural Hemorrhage	G
11	7.2	6000	P_{68} L_{32}	12	4.0	2.0	Microcytic Hypochromic	N	22	100	0.8	N	14	28	NEG	NR	ND	Delta Sign	+	+	+	-	-	Minimal Subarachnoid Hemorrhage over Tentorium Cerebelli	G
12	13.0	7000	P_{70} L_{30}	10	3.6	2.0	N	N	19	98	1.0	N	15	26	NEG	NR	ND	Hemorrhagic Infarct L Temporal	-	+	-	-	-	-	G

SNo.	Complete Hemogram																	Magnetic Resonance Venogram						
	Hb g%	TC/cu mm	DC	ESR mm/hr	RBC mil/cu mm	Platelets lac/cu mm	Peripheral Smear	Urine	Blood Urea md/dl	Blood Sugar mg/dl	Serum Creatinine mg/dl	LFT	PT	aPTT	HIV	VDRL	CSF	Cranial CT Plain / Contrast	SSS	TS	SS	CV	DCV	Parenchyma	Prognosis
13	12.0	6600	P_{60} L_{40}	10	3.0	2.5	N	N	21	86	1.0	N	15	25	NEG	NR	ND	Delta Sign with Hemorrhagic Infarct R Parietal	+	-	-	-	-	-	G
14	8.0	5400	P_{70} L_{30}	11	4.1	2.0	Microcytic Hypochromic	N	22	80	0.8	N	13	30	NEG	NR	ND	Hypodensity both Thalamo, Capsulo, Gangionic region (? Deep venous involv)	-	-	-	-	+	-	D
15	12.0	5100	P_{70} L_{30}	10	3.9	2.0	N	N	20	84	0.8	N	15	30	NEG	NR	N	Delta Sign with Hemorrhagic Infarct R Frontal with surrounding Edema	+	+	+	-	-	Hemorrhagic Infarct R Frontal with surrounding Edema	G
16	7.6	5600	P_{70} L_{30}	12	3.5	2.0	Microcytic Hypochromic	N	22	90	1.0	N	15	32	NEG	NR	N	Hemorrhagic Infarct L Frontal	+	-	-	-	-	-	G
17	12.0	7000	P_{60} L_{40}	10	5.0	2.0	N	N	20	88	0.8	N	16	30	NEG	NR	ND	Hemorrhagic Infarct L Frontal	+	-	-	-	-	-	G
18	13.0	7200	P_{66} L_{34}	10	4.0	1.5	N	N	20	101	1.3	N	14	29	NEG	NR	N	Delta Sign	+	+	-	-	-	-	G
19	8.0	6600	P_{72} L_{28}	9	3.6	2.0	Microcytic Hypochromic	N	21	103	1.0	N	15	28	NEG	NR	N	Hemorrhagic Infarct L Parieto Temporal	+	+	-	-	-	-	G
20	13.0	5000	P_{70} L_{28} E_2	10	3.0	1.8	N	N	28	84	1.2	N	13	30	NEG	NR	N	Hemorrhagic Infarct L Parietal	+	-	-	-	-	-	D
21	12.0	5200	P_{70} L_{30}	13	4.9	2.0	N	N	26	90	1.0	N	13	33	NEG	NR	ND	Delta Sign Hemorrhagic Infarct B/L Frontal	+	+	-	-	-	-	D

SNo.	Complete Hemogram																		Magnetic Resonance Venogram						
	Hb g%	TC/cu mm	DC	ESR mm/hr	RBC mil/cu mm	Platelets lac/cu mm	Peripheral Smear	Urine	Blood Urea md/dl	Blood Sugar mg/dl	Serum Creatinine mg/dl	LFT	PT	aPTT	HIV	VDRL	CSF	Cranial CT Plain / Contrast	SSS	TS	SS	CV	DCV	Parenchyma	Prognosis
22	12.0	6100	P_{66} L_{34}	12	4.0	2.0	N	N	20	96	1.4	N	12	34	NEG	NR	N	Normal	-	+	-	-	-	-	G
23	13.0	7000	P_{66} L_{32}	11	4.0	2.0	N	N	27	100	1.0	N	14	30	NEG	MN	N	Hemorrhagic Infarct R Parietal with Surrounding Edema	-	-	-	+	-	Venous Infarct R Fronto Parietal	G
24	13.0	8100	P_{72} L_{28}	11	4.0	2.3	N	N	25	101	0.8	N	15	32	NEG	NR	ND	Normal	+	+	+	-	-	-	G
25	6.6	4600	P_{60} L_{40}	10	5.0	2.5	Microcytic Hypochromic	N	24	88	0.8	N	12	29	NEG	NR	N	Hemorrhagic Infarct L Frontal	-	+	+	-	-	Sub acute Hematoma with surrounding Edema with L Frontal	G
26	13.0	5000	P_{70} L_{30}	10	5.0	2.0	N	N	25	92	1.0	N	13	28	NEG	NR	ND	Hemorrhagic Infarct L Occipito Parietal	-	+	+	-	-	-	G
27	12.0	5800	P_{70} L_{28} E_{2}	9	4.0	2.0	N	N	28	96	0.8	N	15	27	NEG	NR	ND	Delta Sign	+	+	-	-	-	Hemorrhagic Infarct R Parietal	G
28	7.5	5600	P_{60} L_{40}	10	4.0	2.0	Microcytic Hypochromic	N	24	90	0.8	N	15	32	NEG	NR	N	Ill defined Hypo density R Temporal with surrounding Edema	+	+	+	+	-	R Temporal Venous Infarct with surrounding Edema Thrombus ext to IJV	G

SNo.	Complete Hemogram																		Magnetic Resonance Venogram						
	Hb g%	TC/cu mm	DC	ESR mm/hr	RBC mil/cu mm	Platelets lac/cu mm	Peripheral Smear	Urine	Blood Urea md/dl	Blood Sugar mg/dl	Serum Creatinine mg/dl	LFT	PT	aPTT	HIV	VDRL	CSF	Cranial CT Plain / Contrast	SSS	TS	SS	CV	DCV	Parenchyma	Prognosis
29	12.0	7000	P_{70} L_{30}	10	3.5	2.0	N	N	20	92	1.0	N	14	30	NEG	NR	N	Hyper dense lesion with surrounding hypo dense area L Tempro Parietal	-	+	+	-	-	Thrombus ext to IJV, sub acute hematoma L temporal	G
30	12.0	4000	P_{66} L_{34}	12	4.0	3.0	N	N	26	94	1.2	N	12	32	NEG	NR	N	Delta Sign with Venous Infarct Fronto Parietal	+	+	-	-	-	-	G
31	13.0	4900	P_{70} L_{30}	10	4.0	2.0	N	N	30	100	0.8	N	15	29	NEG	NR	ND	Hemorrhagic Infarct R Temporal	+	+	-	-	-	-	G
32	11.8	5000	P_{68} L_{32}	11	3.6	2.0	N	N	28	101	0.8	N	12	27	NEG	NR	ND	Hemorrhagic Infarct L Frontal	+	+	-	-	-	-	G
33	11.5	5200	P_{66} L_{32}	10	3.9	2.0	N	N	20	102	1.0	N	13	27	NEG	NR	ND	Hemorrhagic Infarct R Frontal	+	+	-	-	-	-	G
34	12.1	6000	P_{70} L_{30}	11	3.5	2.1	N	N	24	105	1.2	N	13	29	NEG	NR	N	Hemorrhagic Infarct B/L Fronto Parietal	+	+	-	-	-	Hemorrhagic Infarct B/L Fronto Parietal	G
35	12.2	6100	P_{60} L_{40}	10	4.6	1.8	N	N	22	99	1.3	N	14	28	NEG	NR	ND	Delta Sign	+	-	-	-	-	-	G
36	13.0	4000	P_{68} L_{32}	13	5.0	1.77	N	N	26	88	0.8	N	15	32	NEG	NR	ND	Normal	+	+	+	-	-	MInimal Subarachnoid Hemorrhage both parietal cerebral sulci	G
37	8.0	5200	P_{70} L_{30}	10	4.1	1.9	Microcytic Hypochromic	N	20	100	0.9	N	15	26	NEG	NR	N	Normal	-	+	+	-	-	-	G

SNo.	Complete Hemogram																	Magnetic Resonance Venogram						
	Hb g%	TC/cu mm	DC	ESR mm/hr	RBC mil/cu mm	Platelets lac/cu mm	Peripheral Smear	Urine	Blood Urea md/dl	Blood Sugar mg/dl	Serum Creatinine mg/dl	LFT	PT	aPTT	HIV	VDRL	CSF	Cranial CT Plain / Contrast	SSS	TS	SS	CV	DCV	Parenchyma	Prognosis
38	8.0	5400	P_{77} L_{23}	10	4.0	2.0	Microcytic Hypochromic	N	22	92	1.0	N	14	24	NEG	NR	ND	Delta Sign	+	+	+	-	-	Partial Thrombus in IJV, Galen and Straight Sinus. Venous Infract R Thalamo Parietal	G
39	12.0	5900	P_{80} L_{20}	15	3.9	2.1	N	N	22	89	1.2	N	12	22	NEG	NR	ND	Hemorrhagic Infarct L Frontal	+	+	-	-	-	-	G
40	12.0	6000	P_{70} L_{30}	15	4.0	2.2	N	N	26	86	1.3	N	13	32	NEG	NR	ND	Delta Sign	+	-	-	-	-	-	G
41	12.0	6000	P_{70} L_{30}	12	3.5	2.0	N	N	22	90	0.8	N	15	30	NEG	NR	N	Normal	+	+	+	-	-	-	G
42	13.0	5400	P_{60} L_{40}	10	3.8	1.75	N	N	24	102	103.0	N	14	32	NEG	NR	N	Delta Sign	+	-	-	-	-	-	G
43	12.1	7000	P_{66} L_{34}	10	4	2.1	N	N	26	105	1	N	13	34	NEG	NR	N	Hemorrhagic Infarct L Temporal	-	+	+	-	-	-	G
44	11.8	5900	P_{68} L_{32}	11	3.5	1.9	N	N	22	96	1	N	15	33	NEG	NR	N	Hemorrhagic Infarct R Parietal	+	-	-	-	-	-	G
45	12	5000	P_{70} L_{30}	15	4.2	2	N	N	28	90	1.5	N	15	29	NEG	NR	ND	Delta Sign	+	+	-	-	-	-	G

S.No.	Complete Hemogram																	Magnetic Resonance Venogram						
	Hb g%	TC/ cu mm	DC	ESR mm/hr	RBC mil/ cu mm	Platelets lac/ cu mm	Peripheral Smear	Urine	Blood Urea md/ dl	Blood Sugar mg/ dl	Serum Creatinine mg/ dl	LFT	PT	aPTT	HIV	VDRL	CSF	Cranial CT Plain / Contrast	SSS	TS	SS	CV	DCV	Parenchyma	Prognosis
46	12.6	6200	P_{68} L_{32}	10	3.6	2.2	N	N	30	92	1.5	N	14	28	NEG	NR	ND	Delta Sign	+	+	-	-	-	Venous Infract R Thalmo Parietal with Thrombus in Straight Sinus.	G
47	12	7100	P_{60} L_{40}	12	3.7	2.1	N	N	32	94	0.8	N	13	29	NEG	NR	ND	Hemorrhagic Infarct L Fronto Parietal	+	+	-	-	-	-	G
48	12	7000	P_{70} L_{30}	10	3.5	1.9	N	N	28	95	1	N	12	29	NEG	NR	ND	Normal	+	-	-	-	-	-	G
49	6.8	5000	P_{60} L_{40}	10	4.1	2	Microcytic Hypochromic	N	26	88	1.4	N	15	27	NEG	NR	N	Normal	+	+	+	-	-	-	G
50	7	5500	P_{66} L_{34}	12	4.2	2	Microcytic Hypochromic	N	22	87	1.3	N	15	26	NEG	NR	ND	Delta Sign	+	+	+	-	-	MInimal Subarachnoid Hemorrhage both parietal cerebral sulci	G

* All patients had Focal deficits in form of Hemiparesis
* All patients had visual disturbances in form of blurring of objects
• Non – Puerperal Cases

Normal Values
LFT - SGOT- 0-35 U/L
SGPT- 0-35 U/L
SAP-40-100U/L
S.Bilirubin - 0.3-1mg/dl
S.Proteins - 6-8 gms/dl
Albumin - 3.5-5.5 gm/dl
Globulin - 2.5 gms/dl

CSF
Cells - <5WBC/miL
Protein - 20-50mg/L
Sugar - 40-70 mg/DL
Chloride - 116-122 mmol/L
Pressure - 50-180mmH2O

ABBREVIATIONS IN MASTER CHART

F	-	Focal
GTCS	-	Generalised Tonic Clonic Seizures
RPL	-	Recurrent Pregnancy Loss
PIH	-	Pregnancy Induced Hypertension
LCB	-	Last Child Birth
LFT	-	Liver Function Test
PT	-	Prothrombin Time
aPTT	-	Activated Partial Thromboplastin Time
A	-	Absent
P	-	Present
ND	-	Not Done
SSS	-	Superior Sagittal Sinus
TS	-	Transverse Sinus
SS	-	Sigmoid Sinus
CV	-	Cortical Vein
DCV	-	Deep Cerebral Vein
G	-	Good
D	-	Death